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CLINICAL LECTURES.

GRIPPAL PNEUMONIAS: I.—GRIPPAL PNEUMONIAS FROM A CLINICAL POINT OF VIEW. II.—ACTION OF THE GRIPPAL POISON. III.—THERAPEUTIC INDICATIONS. IV.—TREATMENT.*

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I.—GRIPPAL PNEUMONIAS FROM A CLINICAL POINT OF VIEW.

Gentlemen:—I have told you, based upon microbiological researches, that grippal pneumonias are not different from the fibrinous pneumonias, since in both cases the same pathogenic germ, the pneumococcus, is found. I have likewise said that another microbe, the streptococcus, is frequently met with in all the pneumonias of influenza. Finally, it has been established that grippal infection is the result of microbian associations, and, without taking into consideration the new bacillus of Pfeifer, there have been described in patients suffering from the grippe many pathogenic germs, such as the streptococcus pyogenes, the staphylococcus pyogenes aureus, and the pneumococcus. Undoubtedly, it is to this association of microbes that the troublesome and complex pathology of this peculiar malady is due. Considering the numerous medical opinions which have originated at every new outbreak of the epidemic, I may repeat what an old historian of the 15th century has remarked: "No physician, nor anybody else, has been able to say what the disease is."

Clinical studies have sufficiently revealed to us that influenza is of microbian origin, and as I have already gone over this matter, though in a rapid manner, I deem it of importance now to consider the subject from clinical and therapeutic points of view. In regard to microbicidal medication, this will first claim our attention.

Certainly during the epidemic of the grippe we find fibrinous pneumonias (that is pneumococcic pneumonias, to use the language of to-day) which do not arise directly from the disease in question. Such pneumonias take their regular course, exhibiting well-known symptoms, symptoms which are unnecessary for me to mention. We also have, however, grippal pneumonias which belong essentially to the epidemic affections, (streptococcic pneumonias, if you please) especially pneumonias which clinical studies have made known to us.

You have seen patients presenting all the signs of pulmonary hepatization with more or less dullness over one of the lungs, accompanied with bronchial murmurs, crepitant râles, increased vocal fremitus, a decrease in the bronchophony of the voice and more or less intense dyspnœa. After these physical signs are manifest, fibrinous pneumonia appears under more marked characters, and, nevertheless, there are noticed the following peculiarities: there are no rusty sputa; the expectoration consists of a thin, frothy and scanty fluid, resembling a gummy solution; sometimes the sputa are compact, muco-purulent and absolutely deprived of air. In these instances, bacteriological examination of the sputa has revealed, according to Duponchel, an important peculiarity, that is, the absence of the capsulated diplococci of pneumonia. That is not all. The patient may have pain on the side, but there is no sign of friction which is so characteristic, or he may exhibit some slight and repeated frictional signs similar to those observed in pleurisy. The disease may come on in an insidious manner, so to speak, making itself known only by a feeble pain on the side, a more or less pronounced febrile reaction and by symptoms of dyspnœa. After the eighth day, defervescence takes place, but this is only incomplete and ephemeral. Soon afterwards the fever is re-estab-

*A Clinical Lecture delivered at the "Hôpital [illegible]."

lished, at the same time new pneumonic foci make their appearance at various points of the pulmonary parenchyma. Here we have, in a word, a pneumonia coming on by successive steps, in which the thermal curve is absolutely different from that of common pneumonia, the condition resembling more one of broncho-pneumonia.

We have other pneumonias characterized by a rapid march, soon arriving at the stage of hepatization, accompanied with a sanious and abundant expectoration, which are really *galloping* pneumonias (so named, since 1880, in the thesis of my pupil Doussian),* their whole evolution taking place in the period of from forty-eight hours to three days at the least.

We also have bastard pneumonias so-called, which are observed under the form of pulmonary atelectasis, of a subacute and slow progress, without great elevation of temperature, and which can be borne for many weeks without any therapeutic interference. You have active and rapid pulmonary congestions, in which a true hæmoptysis may lead you to suspect tubercular trouble, but in which there is an absence of the bacillus Koch in the bloody expectoration. If in such instances the temperature is not very high, it may at least reach 41° C., as in two cases described by Doussian, both of which recovered. Again, these pulmonary congestions are characterized, from a stethoscopic point of view, by a diminution or total absence of the vesicular murmur. Respiration is quiet, the expectoration resembles a thick, gummy solution, and to establish the diagnosis of *congestion of the lung*, you have no other signs than the dullness, the increase in the thoracic vibrations and the dyspnœic condition of the patient. Still in another class of cases, both lungs become the seat of numerous compact, crepitant râles which convey to the ear the sensation of a small, fine shower, the whole condition presenting an aspect of a mixture of pulmonary congestion and œdema. Deceived by the absence of respiratory murmurs, you may be induced to give a favorable prognosis; but the congestive wave, so to speak, continues; both lungs become affected from the base to the apex, cyanosis makes its appearance on the lips and on the extremities which become also cold, and to your astonishment your patient is carried away by almost acute asphyxia. This mechanical asphyxia, however, is not always produced by the congestive trouble; it may also be the result of a true *massive pneumonia*, as we to-day term it, and as Nonat so well observed during the epidemic of 1837, and in which he described pseudo-membranous concretions obliterating almost completely the lumen of the bronchial tubes.

The asphyxia may have a different origin, not being due simply to mechanical disturbance as the result of pulmonary obstruction from the congestive condition. The patient may have bronchitis with limited pulmonary congestion, accompanied with large crepitant râles, but without a blowing respiration. The tongue may remain moist, the bodily temperature but little affected, and led by these negative signs, you will not give much attention to the gravity of the prognosis. You notice, however, a condition of dyspnœa, a condition at variance with the comparatively stethoscopic phenomena. Soon afterwards, the respiration becomes embarrassed, the expectoration is difficult or ceases altogether, congestion advances quickly, and asphyxia rapidly kills the patient; or else, as is often the case, death takes place through syncope, preceded, for a few hours, by the symptoms of cardiac collapse. Graves, who has observed these cases, says, and not without reason, that patients succumb to "pulmonary paralysis." These pneumonias or congestion of *bronchoplegic* or *cardioplegic* form, as I call them, attack the nervous system and particularly the functional activity of the vagi.

I have gone so far without saying anything to you about meta-pneumonic purulent pleurisies, pulmonary gangrene, bronchitis with purulent expectoration, and other forms of pulmonary congestions, etc. I will endeavor to show that there is no such thing as a grippal pneumonia, but that we have grippal pneumonias. Clinical studies demonstrate this and bacteriology confirms it. We have congestive pneumonias, inflammatory pneumonias, and suppurative pneumonias: those produced by the pneumococci, and those caused by the streptococci. The grippe raises to the highest point the virulence of all micro-organisms, and this explains the frequency and the constant threatenings, so to speak, of infections secondary to this disease. If all fibrinous pneumonias terminate frequently by gray hepatization, this is because the microbes of suppuration co-exist with the pneumococci, and it may be said, therefore, that all suppurative pneumonias are the result of secondary infection.*

*Doussian:—Contribution à l'étude des formes cliniques et du diagnostic de la grippe (Thèse inaug. de Paris, 1880).

*D. Plaguere (Thèse inaugr. de Paris, 1886).

Many are the varieties of these grippal pneumonias. Potain said to me one day, apropos of an extraordinary case of influenza which I brought to his notice "With this grippe one has to re-study his pathology." The same thing may be said in regard to the therapeutic management of the disease.

II.—THE ACTION OF THE GRIPPAL POISON.

Grippal pneumonias do exist, as I have endeavored to show you by clinical study; they have their own individuality, their peculiarities and their special characteristics. But in the midst of the various and complex assemblage of these pneumonias, there is an important fact which has called the attention of clinicians of all countries and at all times, and that is, the nervous asthenia, the profound adynamia. This nervous asthenia precedes or accompanies all the local manifestations of influenza, and may at the same time continue for weeks and even months. There is one thing that you have observed, and which Graves refers to as a pulmonary paralysis, and which I myself attribute to a sort of inertia or paralysis of the vagi nerves.

Such may by explained, on the one hand, by the production of rapid pulmonary congestion and on the other, by phenomena of bronchial paralysis which I have studied under the name of *bronchoplegia* or else *cardioplegia*, a condition that is apt to produce death through a marked asphyxia, or suddenly (although this exceedingly rare) by an unforeseen cardiac syncope. The pneumogastrics of certain grippal patients, as I have observed during the last epidemic, are undoubtedly greatly affected, proved by the fact that the pulse is slowed in many cases, such forming a singular contrast with the elevation of the bodily temperature. One of the historians of the epidemic of 1837, Landau, has remarked that the pulse, ordinarily large and full in this affection, was sometimes small and slow, and except in two cases in which it rose as high as 86, it never passed 72, and most generally it varied from 60 to 68.

Again, during an attack of influenza the arterial pressure is diminished, the elasticity and contractility of the weakened vessels no longer sustain the heart which, deprived at the same time of its normal influences and vascular supply, is the prey of exhaustion; hence, cardiac collapse. There comes, similarly, a peculiar syndrome, a condition which I am in the habit of speaking of as a fœtal rhythm of the cardiac beat, a condition which I have studied under the name of *embryocarditis*, and which I regard as the precursory sign of approaching dissolution.

The grippal poison not only paralyzes the pneumograstric nerves, it not only threatens the contractility of the bronchi and the heart itself, but it also acts upon the medulla oblongata, which explains the production of certain hyperthermias; hyperthermias incompatible, indeed, with the by no means marked intensity of the local lesions, with the Cheyne-Stokes respiration, or with the post-grippal glycosuria and albuminuria which I have observed.

III.—THERAPEUTIC INDICATIONS.

I have often said, and will now repeat, that in pneumonia the disease is in the lungs, but the danger is in the heart. In grippal pneumonia the danger lies chiefly in the nervous system. It is our knowledge of this fact that must lead us to recognize the therapeutic indications.

In all grippal manifestations we ought not to consider this or that diseased organ and direct to that alone our therapeutic measures. For instance, a simple disturbance of the stomach shows itself. It persists after many weeks notwithstanding all treatment. If in that simple gastric derangement, (which is only a point in your diagnosis) you take nothing in consideration but a diseased stomach or else an intestine in which there is a rapid increase of toxines or micro-organisms, then your therapeutic medication is irrational. Behind this affection, although simple in appearance, there is an alteration of the nervous system, an asthenia difficult to overcome and against which you may wrestle without a chance of success. This is true during an epidemic of influenza, when without fever or any local manifestation, the disease is only characterized by this nervous asthenia to which the term *grippal state* may properly be given. It is this asthenia, without any gastric disturbance, that you try to combat when it is free from all complication. You must not forget that derangement of the stomach is only an element superadded to the nervous asthenia.

The same holds good in regard to grippal pneumonias; but here, the intensity of the inflammatory processes is such, that very often all your attention is directed to the lungs, which permits you to see only a diseased organ when, in fact, the whole system is affected. What can you do against a pneumonic process which must have its own precise cyclical evolution? Nothing, or almost nothing. We no longer recommend the expectorants, kermes mineral, antimony, that

is, all those remedies which depress the heart; nor blood-letting which depresses the forces and which is only indicated in special cases, such as peripneumonic congestion or threatening mechanical asphyxia; nor even blisters, which constitute the chief indication, are now ever employed. If you follow such a practice, employing these medicaments and measures in a systematic manner, from routine, you will only make conditions worse, by adding a bad remedy to a bad disease.

In the course of pneumonia or pulmonary congestion, there is a compensatory organ which will be called upon to wrestle constantly against the enormous disturbance of the circulation, and such an organ is the heart. It is that which, from the very beginning of the disease, must be sustained and strengthened in a systematic manner, without waiting for the organ to become weakened in its functional activity and contractility. You must not forget that in influenza the heart is more markedly affected in its nervous mechanism than in its muscular element. This in view, you must not commit the error which I shall presently point out to you. A pneumonia, more or less diffused, is developed. The heart, apparently unaffected during the first few days, becomes accelerated in its action, the systole is vigorous, the pulse has a vibrating character, and owing to these circumstances you are apt to believe in non-cardiac interference. You do not notice that the heart is acting violently in the struggle, and that as a result of this, exhaustion of it will ensue, from which it will never recover; and that this apparent cardiac force is an index, a proximate cause of its weakness. Such order of things have I observed in many cases.

Following this period of excitation, weakness of the organ ensues, and it is only at this time that you think of administering digitalis, when nothing can be done for an organ whose contractility has been enfeebled, and when diuresis cannot be produced owing to the impermeability of the kidneys, this condition appearing at an advanced period of the disease. Digitalis must be prescribed at the very beginning of the pneumonia. It is this method of treatment which I especially recommend, and which has been adopted by other authors, such as Traube, Hirtz, Coblentz, Duclos, Sancerotte, Jurgensen, etc.

To combat the effects of pneumonia, or to prevent them, we must direct our attention to the heart, and this should constitute a principle of therapeutics, to which I have given the name of *compensatory therapeutics*. I will explain myself.

You are in the presence of a serious case of icterus. The symptoms become more and more alaming, the adynamia is profound, and hæmorrhages threaten the life of the patient, when suddenly there appears a marked urinary crisis and a fatal issue is warded off. What is the lesson taught us by a clinical case of this nature? It shows us that such a result has not depended upon the liver, that is, upon the affected organ, but upon the compensatory organ, upon the kidneys that serve to eliminate the morbific elements, the toxines which the hepatic cells, profoundly altered, have not been able to destroy or neutralize. You know very well that in order to have a healthy functional activity of the organism, when a portion of it fails in its action, another must take its part. One of the functions of the liver is to destroy the numerous toxic bodies that go through its structure, while it is the duty, so to speak, of the kidneys to eliminate poisonous materials. Therefore, in diseases of the liver our medication must be directed to the renal organs; in cases of pneumonia the heart is the focus around which we must concentrate all our attentions. In one word, and as one of my oldest resident physicians, Weber, has fitly remarked: "When an organ is affected we must look after the healthy and compensatory organs." You must, therefore, exercise in therapeutics a sort of turning movement; and you know that often a turning movement will lead you to the accomplishment of a brilliant victory.

The indications for the use of digitalis present themselves almost in all cases of fibrinous pneumonias, not only to combat the febrile element, not to act directly upon the pneumonic process, (which is impossible), but to give the heart a kind of reserve power for the expected struggle, and moreover, to enhance diuresis, thus bringing about a depuration of the organism. It is to insure this depuration that we must exercise an influence upon the kidneys and upon the digestive tract to modify fermentation by means of intestinal antisepsis.

Finally, clinical studies show that the nervous system must be sustained and strengthened. The administration of digitalis must be resorted to in all these cases of grippal pneumonia in which the vagi nerves and the medulla are more or less affected, and in order also to avoid auto-intoxication through the inactivity of the eliminative organs. Digitalis must be prescribed at the beginning of the disease, on the first or second day at latest, for the reasons I have sufficiently impressed upon you.

IV.—TREATMENT.

In what form and doses shall you prescribe digitalis? It is not well that we should point out the indications for the use a remedy only, it is also of importance to know how to use this, for a good instrument in the hands of the inexperienced becomes a bad one.

I must remind you, firstly, that I have for some time back abandoned the infusion of digitalis. I do not in any way agree with the opinion of Bernheim, of Nancy, who, in speaking of the treatment of pneumonia, says: "I have never seen digitaline act in those cases in which the simple infusion of digitalis has failed, while I have seen this do good in those instances in which digitaline produced no effect." For the last four years I have employed the crystalline form of digitalis in a solution of the strength of 1 to 1000. This preparation is well borne by patients, it has an invariable and certain action, and its dosage is easily managed. I prescribe at once, some time in the day, 30, 40, or 50 drops of the solution mentioned, the last amount representing one milligramme of the crystallized digitaline, or about four milligrammes of the amorphous principle. This dose will appear perhaps somewhat exaggerated, but nevertheless, it has never produced untoward effect, my especial method of administering it having been communicated to the Société de Thérapeutique. I always prescribe it with care, from the beginning, by itself or in conjunction with an exclusive milk diet, this milk diet opening "a way for the action of digitalis." Objection is often raised to the cumulative effects of the drug, to its slow action and elimination, and hence other cardiac remedies have been advocated as substitutes, but I, myself, have never been able to find an equivalent to this marvellous remedy, without which the therapeutics of the heart would be impossible.

It is time to act against exaggerated views, and to proclaim in strong terms that the cumulative effects, the slow action, and tardy elimination of the drug are, so to speak, superadded qualities. I will say now and again that digitalis is a corrective of and provokes diuresis, thus becoming a safeguard, a shield to the organism. It must be given in large quantities, because it is just then that it cumulates and acts slowly. When you desire to put a patient to sleep, you inject into him at once a centigramme of morphia, and not in repeated doses. When you want to reduce a high febrile state, you must prescribe from 1 to 1.5 grammes of quinine at once, and not the same quantities in repeated doses during the day. As is the case with quinine, digitalis and all other slow-acting medicaments are not generally given in fractional doses, since such medicaments are divided in the organism itself, owing to the slowness of their action and their tardy elimination. The method of employment of remedies which are rapidly eliminated, such as the bromides and the iodides, is just contrary to the one we have been just considering. You divide the doses, inasmuch as the system becomes often, and for a considerable time, under a marked influence of the drug. In regard to this point I will cite the case of an epileptic suffering from nocturnal attacks, who received, without any effect whatever, eight grammes of bromide of potassium in two doses in the course of the day, but who was completely relieved by two doses of 2.5 grammes each, one given late in the afternoon, and the other at night before the expected attacks.

Digitalis, therefore, must be prescribed in large doses in order to obtain rapid and good effects. My venerable teacher, Potain, has for a long time observed this method of employment, and I administer, like him, in cardiac affections one milligramme of crystallized digitaline at once. However, this, especially in pneumonia, is a maximum dose, and I would not advise you to go beyond it. I, for my part, will not follow the example of a physician of Bucarest, of Petrescu, who fearlessly uses from six to eight grammes of the infusion of digitalis. The innocuity of such colossal doses cannot be explained, unless we admit a poor quality of the plant employed.

For the next two days, and during which also I administer from 40 to 50 drops of the solution referred to, I do not employ any other remedy, nor even the subcutaneous injections of caffeine, because I am always afraid to give another drug whose effects may lessen or antagonize those of digitalis. Therefore, if the adynamia is profound, if the nervous asthenia marked, I do not hesitate to practise hypodermatic injections of ether or camphor, as these are undoubtedly efficacious against a threatening cardiac collapse. I am in the habit of using the following formula:

℞ Sterilised olive oil.....100 grammes,
Camphor..... 10 "

M. Sig. Inject the contents of a common Pravaz-syringe two to four times a day. These injections are not painful, especially if the liquid be injected as deeply as possible under the skin.

Under the influence of digitaline the ventricular systole is strengthened, the pulse becomes very strong, dyspnœa is lessened, the œdematous congestion of the lungs is averted, the bodily temperature lowered; diuresis is

rapidly established, and during a period of four or five days there is taking place an elimination of the toxic substances produced by the infectious disease. Under the power of the drug the arterial pressure is increased, an important consideration in a malady, which, like the grippe, well characterized, and, as I have shown in my researches, tends to produce vascular depression. If in the course of the malady, the indication makes its appearance again, even after the ingestion of the drug, I do not hesitate to re-administer this after the seventh or eighth day, but then in smaller quantities, about 20 or 30 drops, and without considering the presence of albuminuria a contra-indication as is generally wrongly held. In certain similar cases, digitalis diminishes considerably the amount of albumen, and in the infectious diseases, by provoking diuresis, it tends to remove the toxines, whose retention in the organism would be a serious cause of auto-intoxication.

In conformity with the views held at present, an important element must be taken into consideration in all the infectious diseases. I have already spoken to you of hepatic insufficiency. The liver is, in fact, more or less altered; the hepatic cells do not fully exercise their function in arresting or neutralizing poisonous materials, and these, therefore, find their entrance into the circulation. Yet, an auto-intoxication of the organism may be prevented by the kidneys as compensatory organs of the liver, eliminating a great part of the toxines which this latter organ has been unable to destroy. This explains why at this compensatory period the urine becomes highly poisonous, and why this urinary crisis is, as Buchard has remarked, "one of the safeguards of the organism." But the superactivity of the renal structure has its limits as well as its dangers; the constant passage of these toxic and irritant substances produces at last inflammation of the kidney, and the urine is then charged with albumen. Thus the insufficiency of the renal organs is dependent in these cases upon that of the liver, and we have now an albuminuria caused by a glomerulo-nephritis of infectious origin. You can understand then, that here again the object of the therapeutist would be to bring about a urinary depuration by provoking diuresis, and that the indication calls once more for the use of digitalis.

This hepatic insufficiency which plays an important part, often unrecognized in general and febrile diseases, must be combated by two kinds of medication: The one should be directed to enhance the activity of the liver and the kidneys; the other to remove all sources of auto-poisoning, especially that of the intestinal tract. The first indication is met with by the administration of *milk*, since this article of food is diuretic and through such action the toxicity of the urine is diminished; besides, milk contains sugar capable of being transformed into glycogen, and is likewise poor in salts of potash so often injurious to the economy. It is for this reason also that all food containing salts of potash, such as meat and especially broths, should be suppressed. *Intestinal antisepsis* should then be resorted to, and here comes into play the administration of salicylate of bismuth, naphthol, salol, betol or benzo-naphthol. I prefer the latter medicament in doses of 5 to 6 *cachets* of 0.05 gramme each, and I prefer it on account of its non-poisonous properties, its lack of taste, and of its slight diuretic effects.

Intestinal antisepsis must be rigorously practised in all infectious disorders, and especially in influenza, this pestilential malady *par excellence*, as termed by ancient authors. You must not, however, neglect the practice of *buccal antisepsis*, not only in the case of pneumonia, but even in the apparently least serious manifestations of the disease. Bacteriology has taught us that influenza is the resort of a great number of micro-organisms; and it has also been shown that the malady increases the virulence of such microbes. Finally, we have learnt that secondary infections are quite frequent. Now, we can do little against the primary infection; but we are to a certain extent the masters of secondary infections. In an individual suffering from gastric embarrassment, you will observe, after a few days, the development of a pneumonia which you have not foreseen and which you have no reason to fear. In such a pneumonia, the pulmonary affection follows its natural course, when suddenly all the symptoms are intensified, the expectoration becomes sanious and abundant, and you are finally met with all the signs of a pneumonia which has arrived at the stage of gray hepatization. What has occurred in such a case? A secondary infection has been superadded to the primary one. In this patient suffering from gastric disturbance, the pneumococci, normally found in the buccal cavity, have acquired a great virulence; a secondary infection has alighted, as it were, upon the primary disorder and the microbes of suppuration have rapidly completed their work. During an epidemic, in the course and at the onset of a grippal pneumonia, buccal antisepsis must not be lost sight of, and I have but to repeat here

what I have so often told you. To all your pneumonic patients, held under the grasp of influenza, a malady which markedly diminishes the vital resistance of the organism to the action of pathogenic germs, and which so prodigiously exalts the properties of these, you must order every day, and as often as possible, mouth washes and local applications to the deeper parts of the cavity with solutions of corrosive sublimate. Remember, that the *rôle* of the therapeutist does not only consist in curing (and alas! he does not always cure) but in foreseeing and preventing all complications that may appear threatening secondary infection.

I must yet speak of another medicament, *quinine*, whose employment is generally indicated in all grippal pneumonias, but I do not believe in the preventive properties attributed to the drug by certain authors. I am in the habit of prescribing the hydrobromate of quinine, which I consider superior to the sulphate, especially on account of its sedative effects, in dose of from 1 to 1.5 grammes in the morning, in three doses at intervals of half an hour, and this during the period of three or four days at the most. I have often said that this remedy, like digitalis, must be prescribed in large quantities. I have employed thrice daily *cold baths* as recommended by several authors, but I have not obtained good results. When patients have become agitated, it has not been my rule to follow this measure, energetic it is true, but certainly dangerous in a disease in which the heart is constantly being threatened.

Finally, in those cases in which the injections of camphor, ether and caffein appear to be insufficient to combat the adynamia and the nervous asthenia, I have recourse to the use of the *sulphate of strychnia* in doses of 3 to 4 milligrammes a day, either by the stomach, or according to the following formula:

℞ Distilled water.....10 grammes.
Sulphate of strychnine, 0.01 gramme.
M. Sig. Inject 2 to 4 half-syringefuls in a day.

It becomes evident that you must order this medicament in conjunction with other tonics, such as *Todd's potion*, although this is often abused. I, myself, prefer the *kola* preparations, whose tonic and stimulant properties have been pointed out since 1884, in the thesis of Monnet. The formula which I recommend is as follows:

℞ Tincture of kola.....
Tincture of coca.....aa 60 grammes.
M. Sig. A dessertspoonful, 2 or 4 times a day, in a cup of warm and sweetened milk or in a little curaçao mixed with a little water. This is an agreeable preparation.

These, however, are accessory measures. Digitalis, quinine, the injections of camphor, ether or caffeine, intestinal, and buccal antisepsis, form the basis of the treatment of all grippal pneumonias.

I have not gone beyond the enumeration of all these therapeutic means, and know full well that I am far from having grasped the whole of such a vast subject, and that I have not spoken of the treatment of the complications. But I have endeavored to demonstrate to you that from a clinical, as well as from a therapeutic point of view, we only see in grippal pneumonias a diseased lung; that there is no such thing as an antipneumonic medication; and that we are satisfied with the application of compensatory therapeutics to which I have already referred, that is, by calling upon the healthy and compensatory organs that these may lend a helping hand to the diseased one. I have, in one word, endeavored to impress upon you the truth of this proposition:

In almost all pneumonias, especially in grippal pneumonias, the disease is in the lung, but the danger lies in the heart and nervous system.—Translated from *La Bulletin Medical*, February 14, 1892.

TETANOID CONVULSIONS IN AN INFANT. OPERATION; RECOVERY.

The *Centrall f. klin. Med.* refers to Dr. T-R. Ronaldson's case in which a healthy child, normally born, but with an uncommonly thick umbilical cord remained in perfect health until the ninth day, when on the eleventh day tetanus developed. On examining the umbilicus it was found black in color and very foul smelling. Symptoms of inflammation were not present. The convulsions occurred from time to time. During the intervals the child seemed to be well enough, and was able to take the mother's milk. The stump was washed several times daily with a sublimate solution, and every precaution was used to prevent a recurrence of the attacks without avail. On the twenty-third day the author performed excision of the navel, which was followed by a gradual disappearance of the seizures, they becoming less frequent from day to day, and at the end of the seventh week the attacks had entirely ceased, and the child was perfectly well. There were no micro-organisms found in the excised umbilical stump.—*Edinb. Med. Jour.*

PRURITUS.

℞ Resorcin (medicinal).....part 1.
Glycerin.....parts 2.
Water..... " 30.
M.

TUBERCULAR CARIES OF LUMBAR VERTEBRÆ—HIP DISEASE—MULTIPLE SINUSES—SPONDYLITIS.

By H. AUGUSTUS WILSON, M. D.,

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Delivered at the Jefferson Medical College Hospital.

Gentlemen:—I bring before you this morning the little boy upon whom Professor Forbes operated five weeks ago; a case in which there was a tubercular caries of the lumbar vertebræ primarily, and subsequently a tubercular hip-joint which was excised by some physician outside of the hospital. For some reason which will be explained the sinuses persisted, and on last Saturday I laid open the tracks, removed a portion of necrosed bone, and then stitched up the wound. The case at the time of the operation was of very great interest from the fact that there was a sinus just above Poupart's ligament, which seemed to indicate the presence of a Psoas abscess. On opening the sinus, however, it was found to lead directly to the hip-joint, and was not apparently connected with a Psoas abscess. This is of interest because it shows the various directions the sinuses in hip-disease will take. I now bring the boy before you to show you his present condition. He was taken off of the wire bed upon which he was placed after the operation, a few days ago. The wounds are still healing, but they show the characteristic appearance of tubercular granulations. You will observe on the left side a sinus which continues to ooze, and which is probably connected with a carious spine. The site of the operation is in a satisfactory condition, except at one point. You can all see the extensive cicatrization. The leg is in a good position, and is held so, I think, by bony union. The sinus in front is still discharging a very small amount of bone pus. I think from now on a conservative mode of treatment should be observed, for the sites of the dead bone are so inaccessible that it would be unwise to attempt to remove them. Our hope is that by the curative plan of iodoform emulsion the tubercular bacilli may be killed and the progress of the disease arrested.

TRAUMATIC OSTITIS OF HIP AND SYNOVITIS OF KNEE.

This boy you will remember was in our last clinic. You will doubtless remember his shocking condition. His appetite is now greatly improved; his general health also, and he has no pain. The cicatrization is proceeding satisfactorily at all points. We have obtained primary union throughout the wound except at the point of drainage, which I told you would be kept open to afford egress to the small amount of pus that must continue to form. The small bedsore over the coccyx has yielded to our treatment, which consisted of absolute cleanliness and removal of all pressure by the employment of the cotton ring. You can see the character of the union throughout the extensive incision. There has been a decided increase in the deposit of fat in the tissues, showing that our treatment has relieved the drain upon the system, and enabled him to build up. The temperature chart is decidedly improved, though it still zigzags between 99° and 100°, indicating that there is still some constitutional disturbance. The boy is in a good condition, however, as compared to his former condition, and as he further improves other measures for the relief of his affection will be considered.

SPONDYLITIS.

I propose during the balance of the hour giving an introduction, as I might say, to the subject of Potts' disease of the spine. I find that it is necessary to devote a short time as a preliminary to this important subject in order to make its principles rather more clear to you, so that you may understand its importance when cases are before you. Potts' disease is tubercular ostitis affecting one or more vertebræ tending towards a breaking down of the bodies of the vertebræ, thereby producing a peculiar kink or curvature of the spine. The external manifestations of this curvature are so grossly different from those of rotary lateral curvature that all that is necessary to call your attention to the distinction is to show to you these two specimens. In rotary lateral curvature there is no posterior or angular deflection, but a lateral deflection, manifesting itself by a peculiar bulging of the ribs upon one side and a regular deviation of the line of the spinous process, associated with rotation of the vertebral column. This specimen shows the character of the rotary lateral curvature with an elevation upon one side and a depression upon the other, with a rotation of the spinous processes in such a position that the ribs are thrown backward on one side. In marked

contrast observe the changes in this specimen of spondylitis. Here the bodies of the vertebræ are destroyed, and the weight above has caused a bulging of the spine posteriorly. In Potts' disease there is present what there is not in rotary lateral curvature, and that is a *destruction of the bodies of the vertebræ.* I cannot occupy time now by speaking of the nature of the disease further than to say that it is a primary tubercular ostitis rupturing into the joints and destroying the latter; the bodies of the vertebræ become soft, are pressed upon and yielding produce this deformity. There is no tubercular disease in rotary lateral curvature, while Potts' disease is a true tubercular caries of the bodies of the vertebræ. I have in a previous lecture dwelt upon the subject of tubercular ostitis in general, and as all I then said is true of the condition we are now studying, I will not repeat.

Now for the various names of Potts' disease. Just as in hip-joint disease I found it necessary that you should understand the various names by which the disease was designated, so now it again becomes necessary to call your attention to the synonyms under which Potts' disease is recognized. It was Perceval Potts who first accurately described "Some Forms of Paralyses due to Caries of the Vertebræ." He spoke of the impinging of the vertebral column upon the spinal cord as a result of this caries, and as a result of his elaborate paper his name was applied to the disease, as a tribute to his scientific and practical research. Before that, the affection was called hump- or hunch-back, gibbous spine, etc. Before Potts' time the ordinary bone-setters were practically the only ones who treated this condition, and as a result, the diagnosis was rarely accurately made, and no consideration was paid to the prevention of the deformity or its progress. From the time of Potts' first description in 1779 the treatment has necessarily varied very much. The other names of the disease are spondylitis, from the Latin *spondus*, and *itis*, meaning an inflammation of the vertebra. This term describes the pathology of the affection, and hence is a good name to use. It is also called tubercular caries of the spine. Other names are given to the character of the protuberance. The term *kyphosis* is given to the hump, and signifies merely a hump. Hence this name is rarely used in the study of tubercular caries of the spinal column because it is inadequate. I always deprecate the giving of a man's name to a disease, for the reason that it does not describe the pathological condition which is present. It is generally understood now that Potts' disease means tubercular caries of the spinal column. The correct term I believe is *spondylitis*, because it definitely tells what the disease is.

Now as to the causation of the protuberance. One of the clinical stand-bys in a clinical lecture has been the stick I show here, notched to indicate that the bodies of one or more of the vertebræ have been eaten away. Pressure exerted upon the top of this stick held vertically causes a bending of the column, and a hump is produced. Then, unfortunately, the clinical lecturers go further, and say that a straight stick applied to this bent stick as to a bent tree is the principle of the mechanical treatment of this condition. This is incorrect for mechanical reasons. It would be all right could we get our splints or braces around the spinal column itself, and if the spinal column were composed of one piece as the stick is. In caries we deal with softened ribs and with a softened set of costal cartilages; if pressure be exerted on these alone, there will be produced a yielding as is shown in this specimen. The ribs are pressed forward and the costal cartilages spread. If this will happen without support, will not the same thing follow if we try to bridge up the caries parts alone in the manner indicated? To press on these soft ribs will be to bulge them out without affecting the spinal column whatever. The spinal column is held up and the weight of the head borne not by means of guys of stiff material, but by muscles which pull down. The movements of the spinal column are resisted by muscular effort. In caries of the vertebræ, say in the dorsal region, all of the tissues above that spot, namely, the head and shoulders, are tending to cause kyphosis, and the muscles below are increasing this trouble by contraction. The rational treatment, therefore, would seem to be to remove this pressure. By lifting the superincumbent weight I can overcome the deformity in this stick. If I take this spinal column and put it in the upright position in which there is a curve produced, it is the head and shoulders which produce the curving. If, however, I can put it in such a position that there will be no head weight, there will be no kyphosis produced. Hence, the position of recumbency is necessary in the treatment of this disease, because in that position only is the affected part relieved of the superincumbent weight. I do not say that you must keep the patient in bed to do this, for as the course of the disease runs over two to five years such a procedure would increase the constitutional weakness of the patient. By

mechanical means we can *maintain the position of recumbency* and yet allow the patient to be around gaining the advantage of a out-door life. If you adopt the principle that mechanical support to be thoroughly efficacious must have for its object the mechanical maintenance of the position of recumbency you will have a standard to guide you.

There are three forms of apparatus in use about which I shall speak. *First*, that which is applied to the patient's body below the seat of the disease only. *Secondly*, where a sort of bridging of the diseased portion is attempted. The apparatus supports above as well as below the site of the disease. Here there is a firm foundation and the tissue of the splint prevents the ribs from bending. The *third* form I believe to be the best and most rational, in that its object is the relief of pressure and immobilization, and it therefore maintains the position of recumbency. First and foremost there is, just as is the formation of any architectural structure, a good foundation. In taking the weight of the head and superstructure away from a caries of the spine it is necessary to have a good foundation upon which to rest the apparatus and the load it is to carry. This apparatus is one of the forms employed; it can be made out of leather, felt, steel or other material, must fit thoroughly and comfortably the hips so as to prevent any slipping. Then, having secured a foundation, a couple of steel bands are used to carry the foundation up to the point where the superstructure can be applied. Then the apparatus is used to lift the weight of the head and shoulders from the diseased parts of the column. By means of a minerva or collar the head and chin rest upon the foundation and not upon the spinal column. A jurymast will accomplish the same thing. It consists of a rod carried over the head with a sling applied so that the same object may be accomplished. Thus various methods may be used, but what I wish is that you should understand the principle and ignore the material of which the apparatus is made. In such apparatuses the position of recumbence is maintained for the reason that the spinal column is allowed to maintain its normal position free from weight. There is also a freedom from mobility. If this position is accomplished and maintained, you can have a cure just as you can in any other form of tubercular bone disease. Rest and immobility are necessary in all these cases. Osseous recovery in tubercular-spinal disease can be secured if the case is treated in the proper manner. Other forms of apparatus adopt the crutch form of support. There the weight rests on the axillæ, and the weight of the shoulders only is taken off. It is wonderful how far you can elevate the shoulders while the head sinks down, showing that the weight of the head is not removed. An immense amount of extension of the shoulders can be made before the weight of the head is removed. One of the most difficult things in the treatment of these cases is the securing of a suitable foundation. You can see how small the pelvis is in a child. If necessary the foundation may be extended down the legs in order to secure firmness. One of the methods often resorted to in the treatment of caries of the spine is the application of the plaster of Paris bandage. Professor Louis Sayre, of New York, has rendered a lasting service by the introduction of the plaster of Paris jacket in this disease. He advocates a jacket which is applied by him with perfect satisfaction in most cases. But the trouble is that the jacket has apparently been looked upon as possessing curative properties in itself, and hence has often not been properly employed. Plaster of Paris of itself I need hardly assure you has no curative effect; it only acts by mechanical action alone. Only last week a profound case of Potts' disease was sent to me in which the jacket was so short and loose that it slipped over the pelvis, and the doctor very ingeniously had applied straps over the shoulders to hold it in position. The weight of the apparatus was six pounds, and this was added to the shoulders already bearing too much weight. I could only account for the use of such a contrivance by thinking that the physician was imbued with the idea that the plaster of Paris must have been curative in itself, it certainly did not exert any beneficial mechanical action. Plaster of Paris as used by Professor Sayre is an entirely different matter from the plaster of Paris as used by a large number of physicians. Professor Sayre first suspends the patient so as to secure the position of recumbency. Then he applies first of all a good foundation. He presses in around the waist and on this applies a proper amount of superstructure, and applies it for mechanical reasons to accomplish a mechanical result. We can apply plaster of Paris to a broken forearm in the same manner as it is often applied to many cases of spinal disease, by merely applying the plaster above the fracture and not below. If the apparatus is only carried to the seat of disease we are apt to overlook the fact that the head is an important factor in the development of the deformity.

One of the methods of knowing that your object has been attained is by seeing that the child's symptoms are overcome by your apparatus, and unless this be accomplished you may be sure that its use is harmful or at least is doing no good.

COMMUNICATIONS.

A YEAR'S WORK IN MINOR SURGICAL GYNÆCOLOGY AT THE KENSINGTON HOSPITAL FOR WOMEN.*

By CHARLES P. NOBLE, M D.,

Minor surgical gynæcology, embracing the surgery of the pelvic floor, vagina, bladder, and uterus—excluding hysterectomy—has not received much attention at the hands of recent writers. This is partly because the attention of gynæcologists has been devoted to the marvellous strides which have been made in abdominal surgery, and partly because this field of work is not so brilliant as that of abdominal surgery. It is true that minor gynæcological surgery is not brilliant, but it is very important, and it seems to me that it is in some danger of being neglected by the surgeons of to-day. This fact has led me to report my last year's work in minor surgical gynæcology at the Kensington Hospital for Women, and to make certain observations on the various operations named. The following operations have been done:

Removal of vaginal cyst (post cervical)	1
Clitoridectomy (nymphomania)	1
Excision of carcinomatous nodules (secondary after removal of breast)	3
Uterine polyps (removed)	2
Lipoma (removed)	1
True pelvic abscess (drained after exploratory section had excluded disease of appendages)	1
Atresia of vagina (divided)	1
" " vulva "	1
Cancer of cervix—curretting (death fr m uræmia)	1
Amputation of cervix—cancer, 1; procidentia, 2	3
Anterior colporrhaphy (oval)	1
" " (Stotz)	2
Perinæorrhaphy (Hegar's)	1
" (Emmet's)	17
Trachelorrhaphy	6
Dilatation and curetting	18
	60

One death occurred in a case of advanced carcinoma of the cervix, which was thoroughly curetted by Dr. Kelly. Death followed on the eighth day from "cancerous uræmia." This poor woman had been insane for some weeks—the insanity being due supposedly to absorption of septic matter from the decomposing cancer. The operation was done with the hope of relieving this, and also to get rid of the necrotic mass. The other operations in the report were done by myself. The technique employed was quite similar for all cases. The patient's bowels were well cleared out the day before the operation, and moved by enema the morning of the operation. In addition, the patient had a full bath and a sublimate vaginal douche. Operations were done under ether-anæsthesia in the dorsal position, with the Robb leg-holder and Kelly perineal pad in use. The vulvar region and vagina were well scrubbed with soap and water, the vulvar hair was shaved, and the parts were well douched with sublimate solution. In this way an aseptic field was secured; all operations were done under irrigation with boiled water, sponges not being used. This technique is a little troublesome, but the results secured amply compensate for the outlay of time.

I have made it an invariable rule to *re*-examine all patients before beginning the operation. This can be done most thoroughly when the patient is anæsthetized. If the uterine appendages are found inflamed and adherent, any proposed operation upon the uterus is abandoned. I believe this to be the only safe rule of practice.

For sutures, silk, catgut, and silkworm-gut have been used. For general purposes I like silk; but it should not be used where the sutures cannot be removed in one or two weeks. Catgut I have found very useful for sutures having but little strain to bear, as, for instance, the upper sutures in perineal operations. Silkworm-gut has the advantage that it is non-absorbent; hence it is to be preferred where sutures must be left in a long time, as, for instance, in the cervix, when the cervix and perineum are repaired at the same sitting. It has the disadvantage of being stiff, which property makes it somewhat hard to remove, and gives the patient some pain. After operations the vagina is carefully dried, a pencil of iodoform (25 grains), together with a strip of iodoform gauze, is introduced, the vulva is sprinkled with a powder of iodoform (1 part) and boric acid (7 parts), and then a cotton pad is placed over the vulva—held in place by a T-bandage. For perineal operations the urine is drawn for two days; after which the patient is allowed to urinate. The gauze is removed after forty-eight hours; after which a sublimate douche (1:2,000) is given once daily. The bowels are moved on the second day and regularly thereafter. An abundant soft diet is permitted. The external sutures in perineal operations are removed about the eighth day; the internal sutures a the end of the second week. When the cer

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vix has been repaired at the same time, the cervical sutures are removed at the end of the third week or even later. One should err on the side of leaving the sutures in long rather than that of removing them early.

Patients having perineal operations are permitted to sit up in two weeks; those having a curetting, in three or four days; those having a trachelorrhaphy, in a week, etc.

The secret of success in plastic surgery is good asepsis, and careful, painstaking, and accurate denudation and suturing. I have never failed to secure good union, which has always been primary throughout, with two exceptions—one stitch-hole abscess and one small hæmorrhage (hæmatoma).

DILATATION AND CURETTING.

Within the past ten years professional opinion concerning these operations has fluctuated widely. Before the antiseptic era curetting was considered a dangerous operation. Its danger at that time I feel satisfied was due partly to lack of antiseptic measures, and partly to bad diagnoses. At that time our knowledge of the diagnosis of chronic salpingitis was very imperfect, and many accidents (peritonitis) resulted from operating on the uterus when the tubes contained pus or other septic fluid. Since the antiseptic era in the hands of men capable of making a diagnosis of uncomplicated disease of the uterus, and of excluding chronic pelvic inflammation, these operations have been done with impunity. Of late, the legitimacy of the operations has been questioned by Dr. Joseph Price, on the ground that many cases of salpingitis and pus tubes have come under his care in which dilatation and curetting has been done. This fact is no argument against the legitimacy of the operations, nor against the fact that, when properly done in uncomplicated cases, the operations are perfectly safe and free from danger,

Did the women seen by Dr. Price (and by others, including myself) have the tubal disease before the uterus was dilated or curetted? Were the operations done under rigid asepsis? I believe that blunders in diagnosis and blunders in asepsis should bear the blame in these most unfortunate cases, and not legitimate surgery. In my own hands no such untoward results have occurred. On the contrary, under the strict limitations laid down, my confidence in the value and safety of the operations increases as my experience grows.

DYSMENORRHŒA.

Three cases of dysmenorrhœa, due to partial development of the cervix, with anteflexion, and characterized by "cramps" during the flow, were treated by dilatation. Dilatation in this class of cases has always given good results. The cause of the "cramps" is a poorly developed cervix with a narrow canal, whose calibre is further lessened by the anteflexion.

A broader experience has induced me to use the dilator for dysmenorrhœa much less frequently than formerly. I consider it absolutely contra-indicated if there be tubal inflammation, and believe that it is of little use in relieving pain, unless the latter be distinctly intermittent and cramp-like in character.

The pains accompanying menstruation due to inflammation of the uterine appendages, or of the uterus, or due to a depressed state of the blood, with pelvic neuralgias, are not benefited by dilatation, and in such cases it should not be done.

ENDOMETRITIS.

Fifteen cases of uncomplicated endometritis have been treated by dilatation and curetting. Nine of these were cases of fungoid endometritis with resulting uterine hæmorrhages. I believe that this procedure best meets the indications in all cases of uncomplicated *chronic* endometritis. By removing the thickened portion of the diseased endometrium and providing a freer vent for the uterine secretions, most cases of endometritis can be cured promptly, and the remainder are much improved. The number of cases in which it is necessary to make intra-uterine applications is thus much reduced, and these women are saved the necessity of undergoing a prolonged course of painful intra-uterine treatment. By promptly curing women with chronic endometritis another important point is gained—the disease is cured before it spreads to the tubes.

The results in my hands have been most satisfactory in cases of fungoid endometritis, especially those of short duration, resulting from abortions. Cases of chronic endometritis with purulent leucorrhœa have been most intractable, and in these cases it has been necessary to make weekly applications to the endometrium (by means of the applicator) of pure carbolic acid, Churchill's tincture of iodine, or a saturated solution of chloride of zinc for some weeks *after* the curetting. I wish to call attention to the small number of cases of uncomplicated endometritis in this series. Omitting the fungoid cases, there were 6 out of 128 women admitted to the hospital. This is about the average in my practice.

In fungoid endometritis I have found the curette so valuable, and other methods of treatment (in marked cases) so futile, that I am unable to understand how these gentlemen who oppose the use of the curette treat these cases. The only other resort is electricity; but the curette will accomplish in a few minutes what it requires weeks or even months to accomplish by electricity.

The results obtained by the curette in uncomplicated endometritis are so good that of late, forgetting the teachings of past experience, certain operators have proposed to treat cases of endometritis complicated by tubo-ovarian inflammation in the same way. It seems to me that careful men cannot protest too strongly against such treatment. In the first place, the danger of setting up fresh salpingitis and peritonitis is acknowledged (except by the few) to be great; and in the second place, should the endometritis be cured (which is doubtful because of pelvic congestion kept up by the tube-ovarian inflammation), the graver diseases of the appendages remains. The wiser plan, if the appendages are diseased, is first to remove them, and then actively treat the endometritis; or if the appendages are but slightly diseased and do not require ablation, to treat the patient by applications of iodine to the vaginal vault, and the use of glycerine tampons, at the same time using every measure to improve the local conditions by general medication.

It happens not infrequently that when the inflamed uterine appendages are removed, an endometritis is left which causes the patient some annoyance. These cases are often reported by those hostile to modern surgery as showing that the abdominal section has failed to cure the patient. These gentlemen have a mental strabismus, and do not see that the section has accomplished the end aimed at, the ablation of the diseased uterine appendages. Whether this alone will cure the patient depends upon whether the particular patient has any other disease. If she has an endometritis, this must be cured; if anæmia, or indigestion, or malnutrition, these must be treated.

I wish to protest against the view that endometritis as a rule causes much distress, except the annoyance of leucorrhœa, unless it induces hæmorrhage. Where women having endometritis suffer much pelvic pain, and are semi-invalids, the cause of the pain or invalidism is to be sought elsewhere—in the uterine appendages, or in the vital organs, or blood state. It is a narrow man who attributes all the symptoms complained of by women to disease of the pelvic organs, and who forgets that women have an unstable nervous system, easily influenced by morbid conditions of the general economy.

In discussing endometritis it should not be forgotten that other conditions besides endometritis can cause a discharge from the uterus. Whatever will cause congestion of the uterus will cause uterine discharge. For example, subinvolution, constipation, feeble heart, lazy habits, malnutrition as from phthisis, erotism, etc. Treatment addressed to the causative disorder will stop such uterine discharge. This class of cases calls for no treatment of the endometrium.

STOLZ'S ANTERIOR COLPORRHAPHY.

This operation has been done twice during the time covered by this report. It is especially adapted for cases of procidentia in which cystocele is a marked feature. The cases have been treated with most gratifying success by amputating the cervix above the vaginal junction, doing Stolz's operation on the anterior vaginal wall and Emmet's operation on the perineum, the combination of operations being done at one sitting. I have had but one failure in my experience. This was in the person of a woman having complete procidentia, whose tissues had undergone marked fatty degeneration.

PERINEORRHAPHY.

One Hegar's operation was done on a woman having a patulous introitus. Seventeen operations were done after the manner of Emmet for injuries to the pelvic floor, involving laceration of the levator ani muscle and loss of pelvic support.

It seems strange to me that men who are familiar with the anatomy of the pelvic floor, and with the nature of the injuries it sustains during labor, can differ so widely concerning the nature of the operation required to repair the injuries sustained. Not to consider the lacerations extending into the bowel, there are two general classes of lacerations of the pelvic floor:

1. Lacerations involving the vulvar commissure, and extending scarcely to the vagina, scarcely beyond the plane of the hymen. These are the insignificant lacerations, which at most give rise to a gaping *introitus vaginæ*, but which involve no appreciable loss of pelvic support. Such lacerations are median, and involve no very important muscular structures. The bulbo-cavernosus and the transversus perinei muscles may be divided, but the laceration does not extend far enough up the vagina to reach the levator ani.

Usually it is indifferent whether such lacerations are closed or not. The result to be gained is scarcely sufficient to compensate a woman for submitting to a secondary operation. In my opinion, however, even such lacerations should have a primary perineorrhaphy. If a secondary operation is done, the Hegar operation best meets the indications. The flap-splitting method will answer, but it leaves an ugly fold of tissue at the orifice of the vagina.

2. Lacerations extended along the vagina, and involving more or less of the levator ani muscle or muscles and the deep fascia, according to the extent and depth of the lesion. Such tears, as a rule, not only involve the commissure of the vulva, as in class 1, but also extend up the vagina, aud without exception extended up one or both sulci. They are never median. The tough pelvic fascia seems to deflect the laceration to one or other side. But whatever the explanation, it is a fact that deep, extensive lacerations do not extend up the middle line of the vagina, but up one or both sulci. Sometimes the injury is wholly in the vagina, and the commissure of the vulva remains intact. Hence these tears are Y-shaped when the commissure of the vulva is involved and the tear extends up both sulci. They are Y-shaped when the commissure is involved and the tear extends up one sulcus (one arm of the Y is not represented). And they are V-shaped when both sulci are involved and the commissure escapes (the leg of the Y is not represented). The principal tissues involved in the injury are the levator ani muscle or muscles and the deep fascia. The injury extends two or three inches from the plane of the skin perineum. As is well known, pelvic support depends upon the integrity of the levator ani muscles and the pelvic fasciæ; hence the loss of support resulting from the injury is in direct ratio to its extent. When the lesion is extensive, the bladder, bowel, and uterus prolapse.

A very careful and somewhat extensive study of this subject from the clinical side has convinced me that the foregoing propositions represent the facts in the case, and are not theoretical. I believe that the reason certain gynæcologists of prominence do not accept this pathology is because they do not see much of obstetrics, and they have not studied the nature of lacerations immediately after labor. Anyone can convince himself of the truth of the foregoing statements by studying recent lacerations.

(For the sake of completeness, submucous laceration or over-stretching of the levator ani muscle may be called class 3.)

For the cases embraced under class 2, no operation will yield such results as the Emmet operation. The important lesions are in the sulci of the vagina, and can only be reached by denuding aud suturing the sundered tissues in the sulci. As a means of narrowing the vagina it has the further advantage of making use of the natural folds of that canal. The walls of the vagina fold upon themselves in such way that the lines of contact in transverse sections make the letter H. The sulci of the posterior wall represent the lower half of the legs of the H. It will be noticed that the walls of the sulci lie naturally together, so that, if they are denuded and sutured, the parts come together surface to surface. This is a very positive advantage in securing strong union, in addition to the advantages of raising the posterior wall of the vagina against the anterior, and of making a new *mediate* attachment of the vagina to the levator ani muscles.

I need not dwell upon the advantages possessed by the operation in cases of well-marked rectocele, in which the sulci are very deep. In no other way can the rectocele be so well rolled into the vagina.

My experience with the operation embraces some fifty cases. The results have been uniformly good, with one exception—the case of procidentia already referred to.

The other operations which are recommended for the cure of this class of lacerations are the median operation of Hegar (or some slight modification of it) and the flap-splitting operation of Tait.

Median operations are based upon the theory that the injury sustained is rupture of the perineal body, and that the operation is to restore this body. Time will not permit me to discuss this question *in extenso*. The nature of the injuries has been considered, and they are lateral, not median. The old theory of the nature and function of the perineal body has been disproved by Emmet, whose views I accept; hence it appears to me that the indifferent results secured by median operations are due to the fact that they are based on a false conception of the anatomy and of the injuries of the pelvic floor.

The advantages of the flap-splitting method are even less than those of the method of Hegar. In the rules laid down for performing the flap-splitting operation, we are told not to make the incisions deeper than a half-inch (from the surface of the skin perineum); that is to say, lesions situated further up the vagina than one-half an inch from the skin surface of the perineum are not

affected by this operation. Inasmuch as the entire levator ani muscle lies above this plane, it is evident that the Tait operation is worthless in the class of lacerations under consideration. The Tait operation accomplishes just about what the old Baker-Brown episiorrhaphy did—it narrows the orifice of the vulva.

What I have said about this operation is based upon the anatomical considerations involved and upon the results obtained by a prominent professor in this city. At least twelve patients who have been operated upon by him have been seen by me at the clinic for diseases of women at the Northern Dispensary. All had a narrow vulvar orifice, and all had more or less rectocele, with deficient pelvic support, and the symptoms complained of before operation. My personal experience embraces one case, which was sufficient to demonstrate to me the lack of anatomical basis in this operation.

TRACHELORRHAPHY.

Trachelorrhaphy was done six times. Experience adds to my appreciation of the value of this operation in appropriate cases. I am satisfied that the dangers and failures to achieve good attributed to it by some operators depend upon poor judgment in the selection of cases.

Either insignificant lacerations are repaired or lacerations complicated by inflammation of the uterine appendages are operated upon. In the first case the laceration caused no symptoms, and its repair relieved none; in the second, if the patient escaped a peritonitis as the direct result of the operation, she continued to suffer from the morbid condition of the appendages.

SUPPOSITORIES FOR CHRONIC PROSTATITIS.

For chronic prostatitis, Oberlander recommends the employment of a suppository containing:

℞ Iodoformi.....gr. vij ss–xv.
Solve in
Ol. amygdal. dulcis.....q. s.
Adde
Ol. theobromæ.....q. s.
—ut fiat suppos. no. x.
S. One to be introduced at night following an intestinal evacuation.

The oil of sweet almonds increases the absorbability of iodoform. The proportion of iodoform in each suppository is slowly increased from three-quarters of a grain to a grain and a half. In susceptible individuals larger quantities may occasion toxic symptoms.—*Deutsche med. Wochenschr.*, No. 53, 1891, p. 1435.

ARTIFICIAL FEEDING OF INFANTS.*

By WM. A. TREMAINE, M. D.
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Artificial feeding of infants demands our serious consideration. We have, as Dr. Meigs puts it, two ways of artificial feeding, one empiric, the other scientific, and the physician is compelled every day to choose between the two. If he follows the first method there is a wide latitude, with a possible hope of favorable result at the end. Scientifically considered, it is desirable to approximate the composition of human milk.

As the nursing baby takes milk from its mother it is germ free. This result is easily obtainable. Much more stress has been laid upon the method of sterilization than the preparation of the milk, and we have often left out of sight what is certainly of as much importance as the simple steaming. Milk sterilization is certainly becoming a recognized principle. The method of steaming is probably so familiar to us all that it needs but passing mention. It has been claimed by some writers, that the milk during this process becomes "devitalized," but how this rather vague claim can be established is uncertain. Up to the present date probably the most reliable analyses of mothers' milk are those quoted by Rotch in *Keating's Encyclopedia*, of Forrester, Meigs, Harrington and others. In these tables as a mean result the albuminoids are just at 1 per cent., or possibly at one and two-tenths of one per cent. Dr. Meigs stated nearly ten years ago that human milk never varied much from 1 per cent. of albuminoids, and that he has never seen any reason to recede from this statement. Many other analyses have been made placing the percentage at a much higher rate, but according to the best authorities these analyses are unreliable. In cow's milk albuminoids range from 3 to 4 per cent. Without dispute cow's milk then needs dilution in order to approximate the amount of albuminoids in breast milk. It makes but little difference what the diluent is, providing the dilution is accomplished. Water, barley water, lime water can be used indifferently as regards the formation of the curd.

Mother's milk is slightly alkaline; cow's milk is slightly acid, and personal experience has taught me that the degree of acidity varies much. Between Meigs, Keating and Rotch there has been much difference of

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opinion as to the means to be used for changing this normal cow's milk to resemble the slightly alkaline mother's milk. Dr. Rotch used only ¼ lime water of the total volume to accomplish this result. Meigs recommends ½ of the total volume, and says his clinical experience will bear him out in his statement. Keating very justly remarks that when you consider that in a pint of lime water there are only 11 grains of lime, then two tablespoonfuls would only contain about one-third of a grain. Can this be sufficient to correct undue acidity or to supply lime to the tissues? Roughly stated, Keating's plan is to add to the milk bicarbonate of soda and potash with chloride of sodium in the form of a tablet, the whole to be afterwards steamed. Rotch recommends steaming the mixture first, and adding the lime water.

Does this procedure carry out the principle of antisepsis? We practically add to a supposed sterile fluid a certain quantity of liquid which may have been exposed to the atmosphere for an indefinite time.

The lime water cannot be added before steaming, because the temperature of 212° F. would precipitate the lime. When it is added before steaming, the milk will often turn a light brown or reddish color. This result I have obtained myself where the directions were not perfectly followed. Dr. Harrington explains this as the action of the lime water upon milk-sugar at the steaming temperature. He adds, however, that at the beginning there is a slight alkalinity which disappears entirely at the end of the operation, so that the mixture is rendered sterile but not alkaline. Keating mentions some very interesting experiments regarding the effect of lacto-phosphate of lime on the curd of cow's milk. Briefly stated, he added hydrochloric acid to two portions milk; one containing ½ grain of lacto-phosphate of lime, and the other nothing but the pure milk.

In the first case the resulting curd was smooth and creamy, and in the second case, it was lumpy and irregular. Repeating these tests my experience was unsatisfactory until the cause of failure became evident. Fresh morning's milk tested immediately showed corresponding results, while milk which had been standing all day, and was perceptibly acid, showed but little difference in the curd. This seems to emphasize the caution to sterilize as soon as possible after milking. The source of the milk as we know is of vital importance. Cows fed on brewery grain or silo will yield milk much more acid than those fed in the ordinary way.

One writer gravely remarks that filtered water is used ordinarily in the preparation of lime water, not boiled or distilled water. My experience is that with most apothecaries in this city aqua Pawtuxensis is used to answer all requirements. (Pawtuxet River water is our only supply, and decidedly not germ free.)

Brush, in the *New England Medical Monthly*, questions the use of commercial sugar of milk, and says that one of the faults of the physiological chemist is that he makes no distinction between a substance existing in a natural condition and that substance eliminated and isolated by chemical means. Thus the sugar of milk of commerce and the sugar of milk as it exists in that fluid are regarded by chemists as one and the same thing. This he thinks is not the truth. The sugar of milk in that fluid is all assimilated while the milk sugar of commerce when added to baby food is eliminated both by the kidneys and the bowels. He says he never found sugar present in the urine or fæces of nursing babies, but in three cases of infants fed with mixtures containing commercial milk sugar as recommended in Meigs' mixture he always found sugar in the urine and fæces by Keating's test. He suggests a return to the use of cane sugar. Meigs uses 17½ drachms to eight ounces of the mixture. It would seem as if the amount in Meigs' mixture was rather excessive, but up to the present date the use of milk sugar seems to be the most reliable.

Regarding the fatty matter, the average is about 4 per cent. both in cow's and mother's milk. After dilution the proportion of fat in cow's milk becomes diminished. This proportion must be restored by the addition of some fatty matter, preferably cream. The cream from fancy cattle, Ayrshire or Alderney, is apt to be too rich in fats. The ordinary mixed herd milk is not so apt to vary in percentage. The centrifugal cream of which Dr. Rotch speaks (20 per cent. fat, diluted ¼ or ⅓) is probably available only in the larger cities. Frolowsky has shown that the activity of the stomach's growth is very great in the first quarter of the first year, very slight in the second quarter, and moderate in the last half of the year. The weight of the child seems to be of more importance in calculating the requisite amount of food necessary, rather than the age. One-hundredth of the natal weight plus 1 gramme for each succeeding day for the first month is the rule suggested from the investigations at the Children's Hospital in St. Petersburg, and seems to be recommended by most writers on

this subject. Thus for a baby weighing 7½ pounds you would give one ounce of food and add 15 grains for each succeeding day during the first month. From this basis Rotch devises a schedule giving amounts and intervals of feeding for ages from birth up to ten months. This schedule is subject to great variation. Dr. Rotch cites a case under his care of an infant for six weeks whose general development and weight corresponded to one of 12 weeks, so that it was evident that two ounces of food, the usual allowance of food so far as the age was concerned, was not enough, and its weight indicated a gastric capacity of four ounces which it took and digested easily. In institutions sterilization of milk has gained great credit. In private practice it is not always so easy to bring mothers to a proper understanding of its value and importance. In the first case the patients are under absolute control. Exposure of the milk at the continued high temperature certainly renders it safe as regards germs, thus far we accomplish a great deal. My individual experience has taught me that preparation of the mixture itself as regards alkalinity and tissue formation is equally as important.

DISCUSSION ON PHAGOCYTOSIS AND IMMUNITY.*

At the Pathological Society of London, March 15th, 1892.

By M. ARMAND RUFFER, M.D., Oxon.

When beginning their remarks on immunity and phagocytosis Professor Burdon Sanderson and Dr. E. Klein stated that they could not accept Professor Metschnikoff's views. I will imitate their candor, and define my position in the same manner, by stating at once that I accept Metschnikoff's theory in its entirety.

Not one of the speakers has explained what he means by immunity; and surely this is the first point to decide. Immunity from a given disease means freedom and exemption from that disease. As far as I am aware, there is no animal, whether naturally resistant or protected by artificial means, which cannot be killed by the introduction of a specific microbe, provided a sufficiently large dose be used. To talk of an animal being immune against a given microbe is to speak of something which does not exist, and, although I must confess to having used the word myself, I would in future rather talk of animals naturally resistant and artificially protected.

Now when a naturally-resistant or artificially-protected animal is inoculated with a virus, I believe that the destruction of the micro-organisms in that animal is accomplished by the amœboid cells, which, for the sake of convenience, we will call phagocytes. I am not aware that Metschnikoff, or any of his pupils, ever said that this was the only cause of the increased resistance of such animals. Metschnikoff certainly never said so, but in his writings and in conversation he has simply stated—and indeed proved—that the process of phagocytosis was *one* of the most important factors in the acquired or natural resistance of a given animal against a given micro-organism.

Dr. Woodhead has already stated what we know of the origin of phagocytes, and I would not enter into the discussion of this question were it not that I must make a personal explanation. I have not said anywhere that *all* microphages, epithelioid cells, etc., were derived from leucocytes; what I have said is that in the cases I have observed the macrophages were derived from small lymphocytes. Were any proof necessary that such an origin of plasma or epithelioid cells is of no infrequent occurrence, I would point out that the development of these cells can be seen in the intestines of various animals, where they wander into the epithelium layer; that they have been found in the blood vessels; that when vermilion is injected into the veins of an animal, similar large cells seize on the powder circulating in the blood and carry it into the neighboring tissues—into the medulla of the spleen or alveoli of the lungs, for instance; that no such cells are present in the alveoli of a newly-born guinea-pig until they have been attracted there by some kind of noxa; and, lastly, that in invertebrata the transformation of an amœboid cell into a fixed cell has actually been followed day by day in the living animal.

Let us, however, pass at once to what happens when wandering cells have taken bacilli into their interior; for there can be no doubt that leucocytes have that power, and that the organisms contained in cells undergo some kind of destruction. That is a matter of observation which everyone can notice for himself who possesses a good lens, a drop of frog's lymph, and a few anthrax bacilli.

In the first place, see what happens when a protected or non-protected animal recovers from an infectious disease, or even when it dies after having resisted the malady for a considerable time. In all cases which have

*Continued from issue of Reporter of April 2d.

been studied up to the present, phagocytosis is a well-marked phenomenon. Slight, or even absent, when the animal has no powers of resistance against a given microbe, it increases with a more resistant animal, or when an animal has been artificially protected, and reaches its maximum when the disease is essentially chronic, as in leprosy and tuberculosis. Lately I have been studying human pneumonia from this point of view, and I can safely say that the human lung in this disease exhibits, in their exquisite forms, all the phenomena of phagocytosis.

Dr. Klein, in his able criticism of Metschnikoff's theories, lays special stress on the fact that, in several instances, the process is reversed, and that the leucocytes are destroyed by the bacteria, and not the bacteria by the leucocytes. This he considers a fatal objection to Metschnikoff's theory. As far as I am concerned, I may state that, in almost every one of my papers, I specially mentioned that a large number of amœboid cells perished in the fight. I have shown this to take place in the healthy rabbit's Peyer patch, in the diphtheritic membrane, in actinomycosis, in tubercle, in *charbon symptomatique*, etc.

I will meet Dr. Klein on his own ground, and take as examples two of the very diseases which he himself has mentioned as proving the fallacy of Metschnikoff's theory —namely, leprosy and tubercle. In many particulars these two diseases resemble each other strongly, for both are caused by a hard resistant micro-organism, and, pathologically, both are characterized by the fact that the micro-organisms, whenever present, set up intense inflammation.

Let us make a section through a leprous nodule, and stain it in the usual manner with logwood and carbol-fuschine. I need not enter into the description of the epithelioid and giant cells always to be met with in leprosy, but at once examine the processes taking place in these cells. In some cases such cells, when crammed with bacilli, undoubtedly die, and this death is indicated by the degeneration of their nuclei, the vacuolization of the protoplasm, and the apparent proliferation of the characteristic bacilli in the cells. That is one side of the picture, but let us look at the other side. In some of the cells the bacilli lose some of their definite outline and fuse together into a hard, solid, resistant mass, in which no structure is discernible even when use is made of the most delicate staining reagents and the highest powers at our command. In a later stage this hard resistant mass gradually breaks up and loses its power of retaining staining matter, until nothing is left in the cell but a clear space, which may be either a vacuole, or possibly a kind of membrane with which the bacilli have surrounded themselves. On the one hand, therefore, we can follow the victory of the bacilli over the cells, and, on the other, we have that of the cells over the bacilli. True, in the large majority of cases of leprosy, the bacilli gain the victory, but we are not justified in thinking that the chronic character of the disease, which may last for months and years, is due to the way in which amœboid cells incorporate the bacilli and destroy a large number of them? Unfortunately, the bacillus has means of defence of its own, and thus frustrates the beneficent endeavors of the cells.

It is an admitted fact that in cases of tuberculosis bacilli are often found in epithelioid and giant cells, and there is not the slightest doubt that these cells frequently succumb in the fight. Signs of degeneration are found in microphages, mono-nucleated, epithelioid, and giant cells, with a frequency which can leave no room for doubt. But what is not so easily observed is the other side of the fight, in which the bacilli perish. Metschnikoff has shown that signs of the encapsulation, decay, and death of the bacilli are of the most frequent occurrence in the giant cells of tubercle, and more especially in the giant cells of those animals which exhibit a strong resistance towards the bacillus of tubercle. In man, indeed, such forms of encapsulation are met with in the more chronic forms of tubercle, such as old scrofulous glands or calcified nodules in the lungs. In many cases of tubercle, therefore, the bacilli prove too strong for the amœboid cells, but that in many others the cells are victorious is proved by the frequency with which cured tuberculous lesions are found at the *post-mortem* examination of patients dying from some other disease. As Dr. Klein has said, in these diseases a great many of the leucocytes perish, but he might have added that the destruction of the bacilli is even more marked than that of the cells.

I am willing to allow the accuracy of Dr. Klein's experiment, when, on injecting a quarter or half a syringe of fluid containing anthrax spores or anthrax bacilli, he finds the anthrax bacilli in the blood a few minutes afterwards. Indeed it would be wonderful were it not so, for the frog's phagocytes are able to arrest a certain number of bacilli, but not an immoderate number; they will arrest enough bacilli to kill a mouse, but not the number of micro-organisms

which would kill an elelephant. And similiarly with vermilion. In my experiments I injected one drop of saline solution holding vermilion in suspension, and found that it was absorbed through the agency of leucocytes. Had I injected a quarter or half a cubic centimetre, the vermilion might possibly have penetrated directly into the blood; but I hold that it is not a satisfactory experiment to inject into the lymph cavity of a frog a large quantity of fluid, when the object in view is to study the absorption of solid particles.

In a former paper I have shown that in animals exhibiting some resistance against the bacillus of Chauveau, the disease was strictly limited to the point of inoculation and its immediate neighborhood. When, however, from any cause, physical or chemical, the amœboid cells were prevented from approaching the virus, the disease became general and the animal died. Dr. J. H. Walker and myself have recently obtained one more confirmation of this law, as shown by the following experiments* with another micro-organism.

If an adult guinea-pig be inoculated subcutaneously with 0.25 cubic centimetre of a pure culture of the bacillus pyocyaneus, an abscess slowly forms at the point of inoculation. Twenty-four hours after the injection the point of inoculation is crowded with bacilli and well-filled phagocytes; but—and this is the important point—cultures made with the various internal organs prove absolutely sterile. The bacilli, therefore, are arrested at the point of inoculation by the amœboid cells, and this can be proved by microscopic examination.

Now if at the same time that we inoculate the bacillus pyocyaneus on one side of the body, we inject on the other side a large dose of chloral hydrate, which is yet not sufficient to cause death, and repeat the dose from time to time, so as to keep the animal fully anæsthetized, it invariably dies within forty-eight hours, and all the organs contain the bacilli in incredible numbers.

It is easy to prove that in an anæsthetized animal the leucocytes are inactive. Let us take two small sponges, fill them with a pure culture of the bacillus pyocyaneus, and place each under a guinea-pig's skin. One of these animals we leave as a control, whereas we inject the usual quantity of chloral hydrate under the skin of the other. Six hours afterwards, when we take out both sponges, we find the fluid in the sponge of the chloralized animal almost as clear as when it was introduced. A few leucocytes may be seen in it, ten or twenty at most in one cover-glass preparation, whilst the control sponge is filled with a thick purulent material, which, on microscopic examination proves to consist of innumerable well-laden phagocytes. No wonder, then, that when the leucocytes refuse their work the bacilli should find their way into the tissues. We have vainly sought for any evidence to show that the fluids of chloralized guinea-pigs are better cultivating media for the bacillus pyocyaneus than those of a non-chloralized animal; in both chloralized and non-chloralized animals the bacilli seemed to thrive exceeding well in the fluids of the living body. Here, then, we have another instance of the part taken by phagocytes in the limitation of the disease.

As to the substances which attract the amœboid cells towards the virus, they have been proved to be the poisons secreted by micro-organisms, or contained in their bodies. It is probable also that in some cases these poisons have the opposite effect, namely, that of repelling amœboid cells. I have lately gained considerable evidence to show that the same substance may attract or repel leucocytes according to its state of concentration. I may mention one such experiment. If a small piece of sterilized sponge be soaked in pure turpentine and placed under a guinea-pig's skin, it will be found that, even after a lapse of twelve hours, not a single amœboid cell has penetrated into the sponge. Sections made through the sponge and surrounding parts show that the amœboid cells have emigrated in considerable numbers into the tissues around, but they are arrested at a certain distance from the sponge, and can proceed no further. A piece of normal sponge placed a little distance off is absolutely crammed with amœboid cells, though the turpentine sponge contains none at all.

Now allow a similar sponge to soak in a very dilute solution of turpentine in olive oil, and place this sponge, together with another containing fine sterilized olive oil and a third soaked in pure turpentine, under the skin of a guinea-pig. After four hours the sponge in the solution of turpentine in olive oil is crammed with amœboid cells, whilst the other sponge soaked in olive oil contains but a few, and that in turpentine none at all. Hence, then, we have the same substance exhibiting totally different effects according to the concentration with which it is applied. It is very probable that such is the case with bacterial poisons also,

*These experiments will be related in full at some future date.

and there are already some experiments which support this view. Moreover, as Dr. Woodhead has already pointed out, the same substances which in the non-resistant animal repel amœboid cells, attract these same cells in considerable numbers when the animal has been artificially protected.

When once the poisons secreted by a particular kind of micro-organism have penetrated into the system, it would appear as if in many cases the leucocytes had lost the power of leaving the blood vessels in order to attack the invading micro-organisms. This loss of power is clearly not always due to a paralysis of the cells, as these still emigrate freely in order to attack certain other bacilli, but results from some other cause still shrouded in mystery.

I must refer to Dr. Klein's theory that the cells in the lymphoid tissues attract micro-organisms, and that "the bacteria find in the cells some attractive chemical substance which suits them, and hence these cells, far from being the enemies of the bacteria, are, on the contrary, their places of refuge for a time at any rate. This view seems to me just as feasible as Dr. Ruffer's views that because these cells contain bacteria therefore they are the chief and only bacteria-destroyers. Dr. Klein supports his views by two series of facts, namely, (1) that in a great many diseases the cells are destroyed by the bacteria, an objection which I have already answered; and, (2) that in a great many bacterial diseases the bacteria are found in the lymphoid tissues of certain organs.

In my lecture, previously referred to, I alluded to the fact that when the micro-organisms are inoculated into an animal, the amœboid cells are attracted to the point of inoculation, and that by Dr. Klein's theory it was difficult to account for this phenomenon. In his reply he states that I misunderstood him, and he referred to what takes place in the internal organs. I am sorry that I made this mistake, but nevertheless my argument loses none of its force; for clearly the phenomena at the point of inoculation must be first taken into consideration, and an explanation given which will reconcile Dr. Klein's theory with the phenomena observed. With regard to what takes place in the internal organs, the only evidence in favor of this theory is that micro-organisms are often found in cells. But I contend that this is not sufficient. Should anyone meet a dead lion and find a lamb inside, he, knowing the habits of the lion, would not conclude that the lamb had taken refuge in that. True, after a surfeit of lamb, the lion might die of indigestion, but the chances of the lamb ever getting out alive would be very small. Similarly, knowing the voracious habits of the lymphoid cells of internal organs, before we can accept this theory, observations must be made showing that the bacilli of their own accord force their way into the cells; explanations must also be found for the fact that the micro-organisms perish in the cells in enormous numbers; and lastly, he must explain why, according to Bardach, Sudakewitch, and others, animals normally resistant against a given microbe succumb almost invariably when the spleen is removed.

Remember also that phagocytes exert a distinct choice between two kinds of microbes. They will leave the bacillus of tetanus for the micro-bacillus prodigiosus, and the streptococcus for the bacillus of diphtheria. This is well illustrated in the diphtheritic membrane, where at the surface one can see leucocytes taking in numbers of bacilli, but leaving streptococci almost untouched; with the immediate result that streptococci are often met in the deeper parts of the membrane, and with the remote result that secondary abscesses occurring in the course of diphtheria are never due to the bacillus of diphtheria, but to some other micro-organism.

The study of the diphtheritic membrane shows the remarkable resisting powers of leucocytes, for although the tissues underlying the membrane are necrosed and dead, the leucocytes have the power of marching in their numbers right up to the bacilli and waging war against them, though many, as I have shown, perish in the fight.

Some investigators, the chief of whom is Dr. Buchner, of Munich, have laid great stress on the bactericidal action of the serum of resistant animals. I may perhaps be permitted to express my great admiration for Dr. Buchner's work, and that of his followers, and I am ready to allow that the facts as stated by Dr. Buchner, are in the main correct; but I am far from accepting the view that the fluids of the living animal play any part in the destruction of microbes in the organism.

In the first place, let us see what happens in animals which have undergone no protective treatment. If the bactericidal action of normal serum has anything whatever to do with the resistance of animals towards a particular kind of microbe, then whenever the resistance of an animal against a given microbe is great, the bactericidal action of its serum on that microbe should also be marked; and that when an animal is not re-

sistant, the bactericidal action of its serum should be feeble, or at least slighter than that of a more resistant animal. A rabbit is not resistant, and its serum possesses a strong bactericidal action on the anthrax bacillus; a dog is more resistant, and its serum has no bactericidal action on the anthrax bacillus. We have one exception, that is the white rat, which possesses considerable resistance against anthrax, and the serum of which has a strong bactericidal action on the bacillus anthracis *in vitro*. But this exception proves the rule, for when the bacillus anthracis is inoculated into a white rat, it thrives at first remarkably well, though it is ultimately destroyed by the animal's amœboid cells. That teaches us another thing also, namely, that it is impossible to argue that those phenomena which occur *in vitro* exclusively take place in the living body.

Were other examples necessary, I would draw attention to the fact that, *in vitro*, the serum of the non-resistant rabbit is a bad cultivating medium for the bacillus pyocyaneus, whereas the serum of the resistant guinea-pig is an excellent cultivating medium for the same bacillus. And conversely, I would point out that the serum of the resistant rabbit is an excellent cultivating medium for the bacillus of Chauveau, and that of the non-resistant guinea-pig a very bad medium for the same micro-organism. Moreover, I have proved that the fluids of both kinds of animals when alive form an excellent cultivating medium for this same bacillus. And yet another example of a similar nature. The bacillus of diphtheria in most animals is only found at the seat of inoculation, and in rabbits and guinea-pigs never spreads any further; and yet the serum of such animals is an excellent cultivating medium for the bacillus of diphtheria.

With regard to Sanarelli's experiments with the cell-free lymph obtained from the frog's lymphatic sac, I must acknowledge that such experiments are extremely interesting, and throw great credit on the scientist's ingenuity; but I contend that, from the point of view which interests us, they prove absolutely nothing.

This gentlemen placed particles of organs of an animal dead of anthrax in a small closed collodion bag, from 3 to 4 centimetres in length, and capable of holding about 1 cubic centimetre, and inserted the whole in the lymph sac of a frog. Three or four days afterward he removed this bag, and placed it under the skin of another frog; and then, after a like period, under the skin of a third frog. Examining its contents on the eighth or tenth day after its introduction into the lymph sac of the first animal, he found that no leucocytes had penetrated into the bag, but that the anthrax bacilli were mostly degenerated, and no longer capable of reproducing the disease when inoculated into a non-resistant animal. Now, it is almost impossible to introduce a bag measuring more than one inch under the skin of a small animal like a frog without producing not only serious lesions, but without grave risks of contamination also. In Sanarelli's paper there is nothing to show that the fluid bathing the outside of this bag was aseptic. Moreover, when we remember how leuco-cytes will force their way in a few days into such hard resistant objects as bone and ivory, it is difficult to believe that the inside of these thin collodion bags should have been free of leucocytes. But granting the fluid outside the bag was completely aseptic, and that no leucocytes had penetrated into the bag, I do not allow for one moment that the lymph contained therein was normal, such as that found in living animals.

The lymph during the last days of the experiment, was a mixture of three different kinds of lymph, collected during eight or ten days from three different frogs. Were any evidence necessary to show that this lymph was quite abnormal, I would point to Sanarelli's own paper, in which he says that the histological elements of the tissue introduced into the bag were so altered that they were perfectly unrecognizable. Would anyone maintain that normal lymph will destroy such a hard resisting element as the nucleus of a cell?

Interesting, therefore, as are Sanarelli's experiments, they prove nothing as to the bactericidal action of the normal lymph of a frog.

It has been proved experimentally that after an animal has been artificially protected against a given infectious disease, the serum of such an animal *in vitro* is a bad cultivating medium for the micro-organism causing the malady, and by some this has been set down as a general rule. This is not so however, for the serum of animals artificially protected against the bacillus of diphtheria possesses no bactericidal action on the bacillus of that disease, and the same remark holds good for tetanus. As I was writing this paper I received a copy of a paper by Mossig, in which he states that the serum of rabbits artificially protected against the pneumococcus is a far better cultivating medium for the pneumococcus than the serum of a normal animal. This bacter-

icidal power of serum is therefore not an essential corollary of the increased resistance of artificially protected animals. Moreover, our knowledge of it is at present extremely unsatisfactory, for there is no evidence to show whether it be a specific power of the serum on the micro-organism producing the disease, and on that micro-organism only; and we have no knowledge as to how long the serum of the animal retains this bactericidal power. I have very strong reasons for thinking that it is only of a very transitory nature, passing off long before the animal has lost its increased resistance.

However that may be, see what happens when an animal is inoculated with a micro-organism on which its serum exerts its bactericidal action *in vitro*. For I repeat it, what we want to know is not what happens when a micro-organism is introduced into the test tube together with that wholly artificial substance called serum, but the point is, what happens when it is introduced into the living tissues and blood of an artificially protected animal. I may perhaps be pardoned if I again refer to a series of experiments which Dr. J. H. Walker and I made on guinea-pigs, the resistance of which had been strengthened by a previous inoculation of the bacillus pyocyaneus. We chose this bacillus because my friends MM. Charrin and Roger have shown that the blood of animals artificially protected against this micro-organism possesses strong bactericidal powers.

The guinea-pigs, which naturally possessed a high degree of resistance against this bacillus, had been further protected by an inoculation of 0.25 cubic centimetre or 0.50 cubic centimetre of a virulent culture of bacillus pyocyaneus, and at the time we used them for our experiments they were perfectly recovered and proved to be efficiently protected against this bacillus. The skin of the animals was carefully shaved and two drops of a bouillon culture were injected under the skin. This dose does not produce the slightest symptom in protected animals. The first guinea-pig was killed by chloroform *20 minutes* after the operation, and curve-glass preparations were made from the seat of the puncture and stained with Leoffler's blue. In each slide several leucocytes were found already crammed with bacilli, while a great many micro-organisms were floating in the exudation fluid, and showed not the slightest sign of degeneration. Cultures made from the point of inoculation gave an abundant crop of perfectly healthy and normal bacilli.

Another animal was killed after two hours. Here the leucocytes were already in far greater numbers and contained numerous bacilli. The micro-organisms floating free in the exudation fluid were absolutely normal, and grew abundantly on agar, gelatine and bouillon.

Another guinea-pig was examined after six hours, and the point of inoculation was full of multinucleated amœboid cells, every one of which was crammed with bacilli. A large number of organisms were still free, and consisted of short, straight, and extremely motile organisms which grew abundantly on various cultivating media.

The fourth guinea-pig was killed after ten hours, and presented much the same appearance as the last; whilst in one killed twenty-four hours after the inoculation the leucocytes had emigrated in incredible numbers and the number of bacilli floating freely in the exudation liquid had correspondingly decreased, although such of them as remained free were marvellously active. A culture was made from the point of inoculation, and this proved to contain nothing but bacillus pyocyaneus.

Although the bacilli had remained for twenty-four hours in the living fluids of this doubly-resistant guinea-pig it was impossible to detect the slightest sign that the fluids of the living animal had exerted their vaunted bactericidal action. On the contrary, they proved excellent cultivating media for the organisms.

Further, we found that the cultures made with the bacillus pyocyaneus, which had lived for twenty-four hours under the skin of a guinea-pig proved excessively virulent; and in one case, of a rabbit artificially protected and inoculated straight into the veins, we found active and virulent bacilli eight days after the inoculation.

Some observers object to the use of filter paper for wrapping up the virus when the object in view is to study the bactericidal action of the fluids of living animals on the grounds that it may arrest some of the bactericidal constituents of the lymph. It is strange that these gentlemen should bring forward this argument, as they accept the evidence based on serum which has been filtered through a porcelain filter, and it has been the boast of some observers that the bactericidal action could not possibly be due to the cells contained in it, as the bactericidal action was not abolished by passing through porcelain. In fact, these gentlemen strain at the gnat but willingly swallow the camel. As a matter of fact, however, I showed that leucocytes in few hours had made their way through the

filter paper into the interior of the bags, and it is clearly impossible to suppose that the same paper which is unable to arrest leucocytes should prove an effectual barrier to a chemical substance.

The increase of virulence in the tissues of artificially protected animals is a result which might have been foretold; for, if any micro-organisms survive at all in such animals, they must be those which, from some cause or other, possess qualities enabling them to resist the attacks of leucocytes, and to adapt themselves to their new medium. A short time after the inoculation, all the weaker micro-organisms must have been destroyed whilst only the stronger ones survived, and by this process of elimination a more hardy race of bacilli must have been obtained. And do we not see the virulence of anthrax increased by passing through resistant animals, such as dogs and pigeons, the bacillus Chauveau rendered more virulent by living in the subcutaneous tissues of resistant animals? But I will leave this point, as most probably Dr. Cartwright Wood will shortly have more to say on this subject, though I must again insist on the fact that in a large number of bacterial diseases, if not in all, there is no evidence to show that the fluids of a living resistant animal have the slightest attenuating action on the micro-organisms causing the disease.

I think there are few pathologists who will not readily confess to the feeling of intense astonishment which seized them when they first read Ogata and Jashara's paper, or rather the account of it as it appeared in the *Centralblatt f. Backteriologie.* Everyone had been trying to find a remedy against anthrax, and everyone had failed; and now it was sufficient to inject the minutest drop of a resistant animal's blood under the skin of another animal to cure it of anthrax. True, there were a few points in the experiment which did not appear quite clear, but still the evidence seemed fairly conclusive. Unfortunately, when other experimenters tried to reproduce these results they failed without an exception. But, for the sake of argument, we will suppose that Messrs. Ogata and Jasuhara's results are correct, and that for some reason or other the other experimenters were wrong. Granting all this, even then I do not see how Dr. Klein's contention holds good that the cure of anthrax in cases may be due to the antiseptic powers of the injected blood.

In the first place the quantity injected was infinitesimally small, and it was not always injected on the side where the virus was introduced but occasionally on the opposite side; so that this drop of dog's or frog's serum, before it had the opportunity of acting, was diluted in the whole of the blood of a mouse inoculated with anthrax. Moreover, when we come to see what is the action of dog's blood on anthrax bacilli, we find that it has only a very weak, if any, bactericidal power; so that, if Dr. Klein's opinion be correct, we must suppose that dog's blood, which has normally no bactericidal power, when injected into a mouse suddenly exerts —for reasons not explained—such a strong antiseptic action that it is able to kill millions of bacilli introduced into the mouse's system, although diluted in the whole of the animal's blood.

And what I have said of these experiments applies with even greater force to experiment made with the serum of animals artificially protected against tetanus, which possesses no bactericidal action on the bacillus of tetanus.

Of Mr. Hankin's experiments with the proteid he obtained from the spleen of white rats, I cannot speak from the chemical point of view, but one thing seems to me certain, namely, that this action of this proteid cannot be due to any bactericidal powers it may possess, for it is impossible to believe that a dose of albuminoid matter which has the power of killing a limited number only of spores *in vitro*, should be able to destroy the millions of bacilli inroduced into the excellent cultivating medium formed by the mouse's tissues and fluids. May I incidentally enter a protest against the crowd of new names with which Mr. Hankin has lately attempted to saddle our bacteriological vocabulary? To speak of alexines is scarcely scientific when their very existence is a matter of considerable doubt; but to talk of mycosozin or mycophylaxin when we have not the remotest idea what sozins or phylaxins are, or whether there are any such bodies, is hardly pardonable. Surely the first thing to do is not to give a hypothetical body a high-sounding name, but to isolate and study that body.

Truly the bactericidal properties of the serum of artificially protected animals are suggestive, and deserve to be studied, but I maintain that there is no causal relation between the bactericidal properties of the serum of a given animal against a given micro-organism and the acquired resistance against the same microbe.

One of the speakers has referred to the experiments of Behring, Kitasato, and others, in which the blood of an animal artificially protected against a given microbe possesses the

property of destroying the poisons of that microbe, not only *in vitro*, but also in the tissues of the living animal; and some have gone so far as to say that it was to this poison-destroying power of the serum that the increased resistance of such animals was due. Far be it from me to depreciate in any way the important results obtained by Messrs. Behring and Kitasato, but at the same time it is evident that this poison-destroying power of the blood is not a necessary accompaniment of the increased resistance of such animals. Thus fowls possess considerable resistance, not only against the bacillus of tetanus, but even against the poisons secreted by this bacillus; and yet the blood of fowls possesses no action whatever on the tetanus poisons. Let us turn now to animals artificially protected. Undoubtedly the serum of a rabbit protected against tetanus often possesses the power of destroying the tetanus poisons, but this is by no means *always* the case; and yet the rabbits, whose serum does not destroy the toxines, possess an increased resistance against the bacillus.

We must conclude, therefore, that, although in artificially-protected animals this property of the serum may be noticed, yet it may be absent even in protected animals, and is generally wanting in animals naturally resistant against tetanus; and also that the increased resistance of such animals is not primarily dependent on the poison-destroying power of the serum and tissues.

Another point which has been mentioned is that animals artificially protected against a living microbe can also to some extent resist the poisons of that microbe. Undoubtedly this is occasionally the case, but it is by no means a general rule, nor is this property of being able to resist poisons a necessary corollary of the increased resistance of animals artificially protected. Quite the reverse, in fact; for in some cases artificially-protected animals have less resistance towards the poisons than non-protected animals of the same species. It is occasionally possible to render animals insusceptible to the poisons of the vibrio Metschnikovi, but usually those animals which are already resistant against the vibrio succumb to the action of poisons more easily than non-resistant animals.

Similarly with the bacillus pyocyaneus. Some observers have occasionally been able to protect animals against the poisons of the bacillus pyocyaneus, but in most cases they have failed, although the animals had gained an increased power of resistance. I found, for instance, that by injecting small doses of sterilized cultures at varying intervals the animals invariably died, whilst a few small doses markedly increased their resistance. The conclusion to be drawn, therefore, is that the power of resisting bacterial poisons is not a necessary accompaniment of immunity, but may be present or absent without apparently influencing in any way the resistance of animals.

There is only one class of cells which appear to possess the power of quickly becoming accustomed to bacterial poisons, namely, the cells which are designated under the term of phagocytes. No doubt means will be found to increase the resistance of other cells of the body to bacterial poisons, and far be it from me to say that such an increased resistance and the poison-destroying power of the blood and tissues of the body do not play a part in reinforcing the resistance of animals; but I maintain that at first this property is not a necessary accompaniment of the increased resistance of animals.

It has been proved that the injections of bacterial poisons so influence the cells of the body that, long after their introduction, marked changes take place in the various organs of the body—changes which often prove fatal to the animals. But no doubt the converse is true, and in other cases the changes are of such a nature that they are of assistance to the cells in resisting the action of micro-organisms and their poisons. That is a notion which is as old as the notion of micro-organisms as essential factors in the production of disease.

There are many other points which I would have liked to touch upon, more especially the evidence on the so-called immunity produced by the blood of so-called immune animals. I must leave this for a future occasion, but I may state at once that these experiments, should they prove correct, are capable of quite another explanation than that given by their authors. And may I remind the Society that, more than two years ago, Dr. Charrin and I proved the presence of the poisons of the bacillus pyocyaneus in the urine of artificially-protected animals fourteen days after these poisons had been introduced; and also that we succeeded in rendering rabbits resistant by the injection of the blood of animals suffering from the pyocyanic disease?

The explanation we gave of this phenomenon was a good deal simpler than that now offered us; but we forgot to coin half-a dozen Græco-Latin words in connection with it, which circumstance may, perhaps, account for the fact that our discovery has fallen into oblivion.

In conclusion, it is my firm conviction that the only process which has been proved to be essential to the resistance of animals—whether this resistance be natural or obtained by artificial means—is the power that amœboid cells possess of taking into their interior and destroying micro-organisms, and there is overwhelming evidence to show that this process is proportional to the resistance of the animal. Finally, let me again repeat that neither Metschnikoff nor any of his pupils have ever said that this was the only cause of natural or artificial resistance.

MR. BALLANCE.

Mr. Ballance said that the question of immunity had an especial and weighty interest for surgeons, whose daily occupation was the making of wounds, which, if they were not rendered immune against the attacks of micro-organisms, might seriously imperil life. This immunity was attained as the direct outcome of the actions of the living cells and fluids of the neighborhood of the wound, The surgeon could only aid the natural processes by employing means calculated either to prevent the entrance of, or to destroy, or to inhibit the action of, these deleterious things which could thwart the struggle of the injured tissues towards the attainment of immunity. The healing of the body against the more generalized specific diseases was comparable with the processes employed by nature in the healing of wounds. The presence of pathogenic micro-organisms or their poisons in the body constituted, in one sense, really nothing more than the presence of a foreign body, which it was the function of the natural processes to get rid of. The observation, then, of the manner in which nature could absorb and remove foreign material in wounds would to some extent aid us in the study of similar processes when engaged in warfare against disease. He had been assisted by Professor Sherrington in studying the question of repair, and they introduced small Ziegler chambers into the subcutaneous tissue and peritoneal cavity of certain animals. In the chambers, in the tissue around the chambers, as well as in the normal tissue-plasma and normal peritoneal moisture, they found two distinct kinds of cells. The one kind resembled in all respects the leucocyte; the second kind was much larger, and possessed a large oval vesicular nucleus, which stained faintly, and the protoplasm of the cell body was markedly granular. Both these kinds of cells migrated into the chambers, but there was a definite sequence of events—the leucocytes commencing to enter the chamber shortly after it was implanted into the body, whilst the larger corpuscles (which sections of the inflamed tissue around showed to be the daughter cells of the surrounding connective-tissue corpuscles) did not commence to invade the chamber until some eighteen hours had passed. Briefly, the subsequent events observed were a loss of vitality of the small kind of cell and its inclusion in the vacuole of the larger kind. The larger kind, when all available food in the shape of fibrin and blood-corpuscle has disappeared, commenced to lengthen out and spin fibrous tissue. The less nutritious the more inert the foreign body, the sooner did the amœboid tissue cells become fixed.

They were greatly assisted in the interpretation of the appearances seen in the osmic fixed preparations by the processes described by Miss Greenwood in the Phizopoda. Just as in her interesting observations little monads coexisting in the same water as amœba proteus were by it ingested, so the leucocyte became the prey of the amœboid connective-tissue cell. In the chambers we observed giant cells formed by the fusion of connective-tissue cells, or by nuclear multiplication without actual separance of the daughter cells from the parent. These giant cell forms seemed especially to be present when the substance to be absorbed was more resistant than usual. We arrived, then, in this way at the conclusion that the invading leucocyte had only a temporary purpose to fulfil in the healing of a wound. The rush of leucocytes which occurred towards a spot invaded by micro-organisms might be thought of as useful in various ways:—(1) Though of feeble vitality, they might be able to digest and destroy certain of the foreign organisms; (2) by the death of others a ferment might be set free, which might diminish the vitality of some of the bacteria, and thus cause them to be an easier prey to other cells; and (3) abundance of food (in the shape of leucocytes) for the after-coming connective-tissue corpuscles would be associated with the attainment by them of the highest degree of vigor and vitality.

All the main work, then, in wounds, not only with regard to repair, but with reference to the removal of foreign bodies, such as clot, ligatures, micro-organisms, etc., fell upon the connective-tissues cell. Their observations gave no countenance to the old Cohnheim doctrine of the development of leucocytes into connective-cells. The legitimate succession of cells was now a well-established law in pathology, as well as in embryology, and, according to this, tissue was never produced by

substitution of function. No positive proof had yet been furnished that the elements of the blood took any active part in the restoration of lost parts. It did not appear reasonable or logical that such an indifferent cell as the leucocyte should ever become transformed directly into a fixed tissue cell, and it was still more improbable that it should be possessed with such a diverse vegetative capacity as to undergo a transition in one place into a connective-tissue cell, in another into the bone, and still another into muscular fibre. And if leucocytes in the healing of wounds had a subordinate function to fulfil, it was probable that they had a subordinate place in the fight against disease and in the attainment of a state of either natural or acquired immunity. In the formation of an abscess it was not the leucocytosis to the part which arrested the disease, but the wall of connective tissue formed around the infected spot by the aftercoming connective-tissue corpuscles. The speaker then discussed the question of the action tissue cells had upon substances outside themselves, and gave his reasons for believing that these cells furnished to the body fluids their bactericidal power. The death of leucocytes, which was always occurring, might be thought of as setting free into the body-fluids fibrin ferment, and fibrin ferment (if it existed) must be closely allied to Hankin's anti-toxic substance and Wooldridge's tissue fibrinogen. Clearly, whatever was in the fluids of the body must come from the cells. The normal physiological activity of the cells of the body might thus possibly explain the defensive action of the healthy tissues and fluids against bacterial infection. This clearly existed, for to produce disease a definite dose of micro-organisms must be administered. The normal quantity of anti-toxic substance present during any given period might perhaps correspond to the anti-toxic substance present in the food vacuoles *plus* the quantity secreted from the surface of the cells into the surrounding fluid. If, on the other hand, it were proved that living serum and plasma were not bactericidal, it followed that the anti-toxic substance was a weapon only used under conditions of irritation or emergency by the cell. It might be that an increase in the normal physiological activity of the cells, acquired as a habit, might explain, to some extent at least, some of the varieties of immunity. In conclusion, he desired to express the opinion that, however beautiful the researches might be that were conducted by the aid of the microscope alone in revealing more of the activity and life of cells, yet that in this way we should not progress far on the road towards a true knowledge of the intimate pathology of immunity. The problem of immunity was essentially a chemical one, and its solution must be sought in the laboratory of the chemist.

DR. E. H. HANKIN.

Dr. E. H. Hankin said that all previous speakers had taken one or other of the two sides of the question under discussion, but he believed that it was perfectly possible to take a middle course and combine the two opposite theories. There was a good deal of evidence to support the view that if an animal was naturally susceptible, very little phagocytosis occurred, and *vice versâ*; and on the other hand there was a certain amount of evidence that, after immunity had been acquired, the fluids of the body possessed bactericidal power. It was a pity that those who belonged to the school of solidists seemed to imagine that every additional piece of evidence bearing out the truth of the theory of phagocytes was of necessity evidence against the humoral theory, and similar reasoning had been adopted by those of the opposite camp. It appeared to him that the phagocyte theory was much like that put forward to explain the power of the magnet, and like it, though it was not the ultimate explanation, and did not contain the whole truth, had yet been of service, and had led to good results. He desired to discuss in a somewhat polemical way certain criticisms which had been made against his work. It had been asked if there was any evidence that elexin was a definite chemical body, or was it only a hypothetical body supposed to exist in fluids possessing bactericidal powers? He reminded them that elexins were probably proteids, and, like most proteids, were excessively difficult to obtain in a pure state. Many observers had shown that blood-serum possessed a certain power of killing bacteria, and that this substance existed in small quantity, and was probably allied to the fibrin ferment. Some time later Halliburton isolated a body which he called cell globulin β, and which he thought was identical with fibrin ferment. By putting two and two together the speaker came to the conclusion that the proteid of Halliburton was the bacteria killer, and he did his best to obtain this body in a state of purity. Dr. Wright had suggested that these elexins were identical with the tissue-fibrinogen of Wooldridge; but there was much evidence to produce against this view. They had, for instance, exactly opposite effects as to clotting, and he was prepared to give many other reasons as to his belief in the non-

identity of these two bodies. He then dealt at length with the criticisms of his work by the French investigators; they had repeated some of his experiments and obtained different results, but they had not used cultivations of the same virulence as his own. They regarded the death of the micro-organism not as due to its being poisoned by the chemical substances contained in the fluid, but rather to a change of the media. This he considered as tantamount to saying that if a man died of poisoning by arsenic he had succumbed to a change of diet. A baby rat's blood had no power of delaying disease or of preventing it; but yet it had just as great a power of attracting leucocytes to the seat of inoculation as that of an adult rat; this was because the blood of the latter contained a fluid of bactericidal power which was lacking in the former.

DR. SIMS WOODHEAD.

Dr. Sims Woodhead, in reply, drew attention to the word "immunity," which was used in two different senses in clinical and experimental work. A patient was only exposed to the ordinary chances of infection as usually understood, whereas an animal in the hands of the experimenter was subject to very different conditions, as for instance, when injected with an organism of specially virulent character. It was well known that under suitable conditions an organism might become much more virulent than usual. In the case of the tubercle bacillus, if it were injected into the peritoneal cavity of an animal, which was ordinarily insusceptible to the bacillus, tuberculosis could be produced with great certainty. He thought that specificity was to a great extent a question of phagocytosis; that, if an animal was immune, it was due to its phagocytic power. Professor Klein's case of injecting innumerable anthrax bacilli into the lymph-sac of a frog had no analogy, and, as Metschnikoff had stated, the phagocytic power of the leucocytes might in certain individuals be imperfectly developed. With regard to the main question, all the positive evidence was in favor of phagocytosis so far as the the earlier stages of infective diseases were concerned, and it was the only theory on which both the experimental and clinical evidence could be explained. There was no real evidence for the humoral theory, and such would always be difficult to get. Results obtained with serum, which was a product of dead blood, could not be assumed to hold good for living blood. He quoted some experiments of Pflüger's, in which, after injection of peroxide of hydrogen into the blood of a living rabbit, no effect was produced so long as no blood escaped; but, if a single drop exuded, an evolution of gas occurred, not only in that drop, but throughout the blood of the animal. Again, freshly drawn pus had bactericidal properties which disappeared after the pus had stood for some time. These experiments showed how delicate was the substance with which we were dealing. Klein's fluid, obtained by dialysis through collodion from the lymph-sac of a frog ought not to be considered as exactly the same as the lymph from which it was drawn; yet results obtained with this fluid were taken as holding good for the fluids of the living animal so far as its bactericidal powers were concerned. He could not agree with Klein's statement, that bacilli got into the leucocytes to avoid the bactericidal blood-plasma, for many of them were non-motile, and could not possibly push their way into the leucocytes. If a paralyzed lamb disappeared in the neighborhood of a lion, it would be only fair to accuse the latter of some active part in the transaction. He firmly believed that the discussion, if it had not brought to light much new truth, would yet open up many new paths for investigation.

SIR GEORGE HUMPHREY.

Sir George Humphrey, having thanked those who had contributed to the discussion, congratulated England on its rising school of bacteriologists, who had accomplished so much, though having great disadvantages to contend against, owing to the false sentimentalism and prejudices against the true interests of humanity which existed in that country. Though he expressed himself as unable to sum up at all adequately the points of the discussion, yet he desired to offer the impressions the remarks of the speakers had made on his own mind. Zymotic diseases were dependent upon the introduction of micro-organisms into the system, and these microbes possessed the power of rapid propagation, and exerted a pernicious effect upon the organs of the body. They produced, moreover, certain excreta or toxines, which, even more than the microbes themselves, were prejudicial. The microbes and their toxines could not be dissociated; they both came in contact with the cells of the body, and with the body fluids. These body cells were undergoing continual change—change essential to their vitality, absorbing, modifying the material absorbed, and returning what was excreted into the fluids around. All body cells were phagocytes; that was an essential feature of their existence. But they were promiscuous feed-

ers, and would take up other things besides those normally supplied from the blood—they would absorb foreign matters—they would certainly absorb bacteria. Speaking technically, they were phagocytic as regarded the bacteria with which they came in contact, and there was abundant microscopical evidence of this, that they could exercise a solvent or destructive influence upon them. By far the majority maintained that the cells were an important element in the destruction of bacteria. Professor Burdon-Sanderson has found it difficult to conceive that there could be any special function in cells by which they could exert this malignant influence upon bacteria. But it was not necessary to assume that they were specially bactericidal; they were only phagocytic to bacteria by virtue of their general phagocytic qualities; it was a part of their general natural function, and not a special endowment. There was evidence that certain poisonous elements introduced into the blood did tend to attract leucocytes into their neighborhood. In some cases, the cells, so attracted, overpowered and destroyed the bacilli; while in the other cases they failed to do so, and the microbes spread over the system. In such instances cells might be found packed with microbes, the latter afflicting the former with such an indigestion as to cause their death.

On the whole, the general result of the discussion seemed to be in favor of the phagocytic action of leucocytes and other cells upon bacteria. A much more difficult question was the effect of the body fluids upon the bacilli and their toxines. In considering that we passed out of the range of ocular inspection and almost beyond the range of chemical examination, we had to deal with fluids of very peculiar composition and very great instability. It was found that the fluid serum in the test-tube might be fatal to a microorganism, and yet inject that microbe into an animal from which the serum was obtained, and it would flourish there; this showed with what delicate materials one had to deal. In this part of the discussion it seemed to him that the probability was in favor of the humoralistic view. But the fluids and the solids of the body interacted so intimately each upon the other that there could be no such thing as solidism and humoralism separately. Anything that could affect the blood serum must also produce an effect on the cells and the bacteria in it. The toxine of the bacteria diffused throughout the blood serum must exert some pernicious effect upon that serum as well as on the cells, and the serum so altered might finally react on the bacteria, and we must not assume that there was no effect because we could not demonstrate it. All the results he had spoken of so far have been obtained by experiment upon animals, but Dr. Bristowe had thrown some practical light upon the subject. He had remarked especially upon the resistance offered to the entrance of bacteria to the body, and referred to the probability that they became altered in their passage. When we considered the utterly inappreciable evidences there were to account for the degree of immunity possessed by different persons, we should not be surprised that it was surpassingly difficult to trace out the causes of immunity in different animals. He regarded it as almost hopeless to form an idea of the essential nature of the change which produced immunity from small-pox by vaccination. He regretted that the point of exhaustion had been rather summarily excluded from the debate. The manner in which a disease attacked one part, ceased there, and passed to another was difficult to explain on any view, phagocytic or other. The migration area in the course of a common cold was an instance in point. He referred to the phenomena in scarlatina, its incubation, its sudden accession, the sequence of phenomena to quiet subsidence, and, if not fatal, the immunity of the subject for a period. How was this explained by phagocytosis or any other theory? To what influence did the microbes succumb after having obtained a maximum of power? Was it because of some poison which they themselves engendered, or because they had exhausted the pabulum upon which they existed? It was difficult to explain the course of certain diseases of the skin by any theory of phagocytosis. We might rest assured that the interest in this subject had not yet passed away, for many of these problems were still invested with uncertainty, and that uncertainty was an invitation to work yet to be done.

MORTALITY OF DIPHTHERIA.

The comparative mortality of diphtheria in the principal countries of the world is as follows: England heads the list with only 41 deaths per 100,000 inhabitants. For the same number of inhabitants Belgiun had 44, Holland 53, Switzerland 59, Italy 79, France 80, Germany 100, Scandinavia and Russia 110, Spain 112, Austria-Hungary 116, and America 140. The statistics were collected by M. Janssens, the Chief of the Bureau of Medical Statistics of Brussels.—*Revue Médico-Pharmaceutique*, October, 31, 1891.

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LEADING ARTICLE.

THE RELATION BETWEEN EPILEPSY AND CHOREA.

Scarcely any other two diseases have been so extensively written about and theorized upon, with less discovery of absolute facts regarding them, than epilepsy and chorea. When all the mass of literature concerning them is sifted down, no more can be said than that their pathology is still a matter of uncertainty. Notwithstanding, it is of interest to realize exactly how far these two disorders can be compared, and if enough can be gleaned from our knowledge to connect them with the same causative conditions, something at least will have been gained. With this in view, Dr. G. P. Trowbridge, of the Danville (Pa.) State Hospital for the Insane, has made some careful clinical studies, and has recently published in a late issue of the *Alienist and Neurologist* a very interesting paper discussing at some length the relations which may exist between these two neuroses, as well as giving the results of a collective investigation among other asylums of cases which support the view that a very close relation does exist. Although for for many years the likeness existing between chorea and epilepsy in many of their features has been noticed, yet very little has been written concerning it, and for this reason we are indebted to Dr. Trowbridge for his interesting paper.

In the purposes of his paper, in order to compare cases of these neuroses in as pure a form as possible, Dr. Trowbridge confines his remarks to idiopathic examples of the two affections, excluding all those cases in which discoverable causes existed, although we cannot fully agree with him when he relegates all such cases to a category which he broadly terms "pseudo-forms of the disorders;" for it is in entire accord with our knowledge of these affections, that their real cause may be engendered by *very various* physical causes. Although, therefore, in our opinion, there is no reason why the discussion of the points of similarity between the two neuroses should not include all cases of either disease, no matter what the nature of their

causation, still, since the cardinal features of both are perhaps most strikingly apparent in the large class of unexplainable cases we call *idiopathic* or *essential*, we will confine these remarks to that variety of the two diseases. Clinically, we find the following points of similarity between chorea and epilepsy.

I. Both are markedly hereditary, and very often it is found that they affect the same families throughout several generations, and that choreic parents will have epileptic children.

II. Both diseases are characterized by convulsive movements of the muscular system,—in epilepsy this is periodic (but it sometimes continues for hours and even days), and in chorea it is a constant symptom except during sleep. The convulsions in both are undoubtedly due to interference with the stability of action of the nerve cell, causing them to discharge nerve-force irregularly.

III. They may coexist in the same patient, or a patient may first have chorea and have it cease only to mark the beginning of epilepsy, or epilepsy may usher in chorea.

IV. Both chorea and epilepsy affect the mind to some degree, and it is said by Warrington that the mental disturbances arising from them are very similar, so that it may be said that they produce nearly the same mental affections; and it will be admitted that these forms of insanity do not conform in type to other forms of ordinary insanity.

V. Both diseases are probably due to lesions of the brain only, and both are chronic disorders, or incline to become so.

The above are some of the points of likeness which chorea and epilepsy have in common. In support of the assertion that these diseases sometimes exist simultaneously in the same case, Trowbridge has collected seven such cases from American asylums for the insane, and fifteen cases which were affected with both diseases, but not at the same time. In seven of these fifteen cases chorea appeared first and was followed by epilepsy, and in only three cases did epilepsy appear before the development of chorea. In two cases chorea merged directly into epilepsy, while in the remaining cases an interval elapsed between the cessation of the one and the onset of the other.

Having glanced at one side of the question, let us look for a moment at the reverse side. Does *chorea of childhood* have any close relationship to hereditary epilepsy? If the affections were due to different degrees of the same pathological cause, should we not expect that their percentages of cures would be more nearly the same?

To the contrary we know that chorea of childhood in the large majority of cases runs a more or less definite course and that recovery is the rule and not the exception. Certainly very few rational signs point to any very close relationship between ordinary chorea of childhood and idiopathic epilepsy.

It is different, however, with hereditary chorea and chorea of adults without hereditary predisposition. These choreic manifestations are progressive, always attended by mental disturbance of some sort, and go from bad to worse until death terminates the picture of misery they present. No treatment influences their course or even alleviates the movements. It would seem, therefore, that between those choreic states of later life and chronic epilepsy there is a marked relationship, and that further study may unearth facts which will further prove the truth of this.

Many of the families affected with hereditary chorea have epilepsy very frequently in some branches of the genealogical tree, and sometimes it seems as if it had taken the place of chorea in one generation, which again reoccurred in the next; but too much importance cannot be laid upon this point for the same can be said of phthisis, which has been found to frequently exist to a remarkable degree in families subject to hereditary chorea.

This whole subject is one of purely speculative interest at present, and the most that can be said is that *chronic* forms of chorea present phases of causation, symptomatology, duration and termination which may justly make us consider whether the relationship between them and chronic idiopathic epilepsy is not more than a mere resemblance.

BOOK REVIEWS.

THE PRINCIPLES AND PRACTICE OF MEDICINE. By William Osler, M. D, New York. D. Appleton & Co., 1891.

The accuracy, clearness, and directness, the thoughtfulness and wide knowledge which have made Dr. William Osler's name so well-known to all serious students of medicine as a science, are as agreeably conspicuous in his latest book as in his many previous publications. A wholesome disregard for the "authority" which consists in the repetition of old commonplaces, and too easily accepted theories, is another familiar characteristic to be found in the present as in its author's former work—and the book will surely secure for itself a larger and more appreciative audience than the undergraduate or young students for whose use it is chiefly meant.

The arrangement of subjects is new and practical. First, specific infectious diseases—thirty-five diseases, including some described as doubtful (Well's disease, milk diseases, Malta fever, mountain fever, and sweating sickness), are included; constitutional diseases follow, then diseases of the digestive, respiratory and circulating systems, of the blood, of the kidneys, nervous system and muscles, the intoxications (obesity is construed as one of these), and last, those due to animal parasites.

It is impossible to undertake in a review to point out all the things which interest or strike the attention in a large book, whose subject is the whole science of medicine, but some detailed examination is both a pleasure and a duty here.

In the opening article on typhoid fever an excellent historical account of the progress of the differentiation of that disease from typhus and the other continued fevers is given, a result largely due to Louis and his American pupils, the Philadelphians Gerhard & Stillè, and George C. Shattuck, of Boston. The modes of infection are not considered at very great length, but an account of the instructive epidemic at Plymouth in 1885 is included. We turn to the treatment to find what the author has to say as to the newer methods. A moderate ground is taken as to the Brand treatment, though perhaps too much stress is laid upon the sufferings of patients from the cold bath. The statistics quoted are not strikingly in its favor: "expectant plan" mortality, 14.8 per cent.; incomplete bath treatment, 12.3 per cent.; strict bath treatment, 7 per cent." We had occasion several years since to examine the mortality of typhoid during a period of ten years (before the use of Brand's treatment) in two of the chief hospitals of Philadelphia. In neither did the mortality exceed 11 per cent.

For private practice Prof. Osler recommends in preference a lukewarm bath gradually cooled, or the cold pack, or repeated sponging. He pronounces antiseptic medication of no value. The routine administration of turpentine he thinks "is a useless practice for the perpetuation of which in this generation H. C. Wood is largely responsible."

In malarial fever Dr. Osler insists properly and very strongly on the absolute value of Laverou's hæmatozoa in the case of doubt in the diagnosis. "Not a single observer who has had the necessary training, has failed to demonstrate the existence of these parasites." Another means of attaining certainty, which anyone may apply, is "that an intermittent fever which resists quinine is not malaria." An illustration of the forms of the parasite and a note of the powers required in lenses for searching them would have been useful additions to this section.

As might have been expected, the book is strongest in its description of these affections where the nice observations, trained acuteness and painstaking minuteness of the diagnostician are most required. No such clear and simple presentation of the conditions, rational and objective, and the pathological anatomy of heart diseases, is known to us as is here made. One may occasionally criticise the refinements of pathological language as excessively technical. A student would make little of a statement that "a subvalvular stenosis, the result of endo-carditis in the mitro-sigmoidean sinus, usually occurs as the result of a fœtal endocarditis."

Another especially good chapter is on anæmia. The author nails his banner to the mast and stands for heroic doses of iron, Bland's pill for choice, and for quantity, say eighteen grains a day of the sulphate of iron, "one of the most brilliant instances of the specific action of a remedy." The section devoted to nervous diseases includes the most recently described forms of which modern refinement of diagnosis has added so many, and indeed the whole book is thoroughly up to date.

Dr. Osler is something of a nihilist therapeutically, and there are places where his laudable distrust of drugs carries him too far, but as a rule the treatment, which is

rather outlined than described, is on the whole sufficient. It is far better not to inculcate the sprouting M.D. with the idea that drugs are all powerful and must prevail. Let him have a wholesome distrust of their infallibility, and let him imitate Dr. Osler too in his accuracy and simplicity of thought and word, and in his hearty devotion to the highest and best work of his profession.

LITERARY NOTES.

THE WORLD'S COLUMBIAN EXPOSITION.

Bond & Co., 576 Rookery, Chicago, publish a four hundred page advance Guide to the Exposition, with elegant Engravings of the Grounds and Buildings, Portraits of its leading spirits, and a Map of the City of Chicago; all of the Rules governing the Exposition and Exhibitors, and all information which can be given out in advance of its opening. Also, other Engravings and printed information will be sent you as published. It will be a very valuable Book, and every person should secure a copy.—Price, 50 cents, post-paid.

SOME WAYS OF SERVING SWEETBREAD.

From Harper's Bazar.

Sweetbreads are daily becoming more and more in demand, both to tempt the capricious appetite of convalescing invalids, and for dainty dishes on dinner, lunch, or tea tables.

While by no means difficult to cook, care must be taken to follow the directions for their preparation, the process being the same no matter which of the recipes is chosen.

In selecting, the larger, plumper, and more fleshy they are, the better. Put them first into tepid water, letting them remain from fifteen minutes to half an hour, then set on the fire in cold water to which a little salt has been added. As soon as the water begins to boil, pour it off, and slip them into a pan of cold water until they are perfectly cold. This parboiling and chilling, which is termed *blanching*, makes them fair and white.

Next remove the pipe and skin. For this purpose scissors are better than a knife. (It is said that woman signalized her entrance into the chemist's laboratory by substituting the pitcher and scissors for the bottle and knife always used by man.) And as the sweetbreads are in pairs cut them apart.

Served Whole with White Sauce.—The sauce requires half a cup of milk, one tablespoonful of flour, and a piece of butter the size of an English walnut (allow the flour and the butter to each sweetbread). Braid the flour and the butter together, let the milk come to a boil, stir in the flour and the butter, add a blade of mace, and let all simmer five or ten minutes. The sweetbreads are simmered in this sauce until every tinge of pink disappears. They are kept in shape with the wooden toothpicks—called toothpick skewers—used for fish and small birds.

When thoroughly done, lay them on a dish, put a border of parsley around them, and pour the white sauce over and around them. The mace is, of course, removed before serving.

Sweetbreads with Cauliflower.—Cut the cauliflower into handsome pieces, blanch and trim the sweetbreads, and lay them all in a deep saucepan. Fill two-thirds of the depth of the pan with water, season with a few blades of mace, pepper, and salt. Let the whole stew for three-quarters of an hour.

To serve, place the sweetbreads in the centre of the dish, arrange the cauliflower in a border, and pour the gravy—thickened with cream and a tablespoonful of butter and flour rubbed together—over the sweetbreads.

Sweetbreads with Tomato.—Stew a quart of tomatoes until they are quite smooth—which will require about three-quarters of an hour—then rub them through a strainer so that you will have a smooth thick sauce. Have ready four sweetbreads, blanched and trimmed. Put them into a pan with the tomato, season with salt, pepper, and mace or nutmeg. Add two or three tablespoonfuls of butter rolled in flour. Set the pan over the fire, and stew until the sweetbreads are thoroughly done, which will be in about three-quarters of an hour. Before taking off, stir in the well-beaten yolks of two eggs. Serve with small pieces of fried bread placed in the dish with the sweetbreads, and the sauce poured over.

SAUERKRAUT IN DYSPEPSIA.

Dr. Heller, of Vienna, thinks he has found a remedy for dyspepsia in that well known digestive tonic, sauerkraut. It is especially recommended to nervous invalids who suffer from eructations and flatulence after meals. Dr. Heller is not trying to be funny, but apparently means what he says. That may do for the German stomach, but, perhaps, the American dyspeptic would do as well to stick to mince-pie and hot soda biscuits, with an occasional Welsh-rarebit thrown in.—*Med. Rec.*

PERISCOPE.

THERAPEUTICS.

CORN CURES.

Professor Whelpley gives some interesting facts in *Notes on New Remedies* about corn cures. The sale of plasters and other applications for corns is a large business, and all these preparations have as their active basis salicylic acid. It is combined with collodion and 9 to 48 parts, and painted over the corn; it is combined with rosin and balsam fir, with lanolin, 10 drachms of lanolin to 6 salicylic acid; with simple cerate, one of the acid to six of the cerate, and so on.

THE BROMIDES AND INCREASED SUSCEPTIBILITY TO INFECTION.

In the *Mercredi Médicale*, October 21, 1891, Dr. Féré takes up the assertion that patients undergoing bromide treatment are more susceptible to infection than others, particularly to the influence of Koch's bacillus. Upon this latter point there are no positive clinical evidences within the author's personal observation, yet during epidemics of pneumonia at the Bicêtre those persons under the influence of bromides proved most vulnerable. Animals inoculated with tuberculine virus after large doses of bromide began to lose ground immediately, and soon died.

ELECTROLYSIS OF THE CEREBRAL CORTEX IN THE TREATMENT OF JACKSONIAN EPILEPSY.

Dr. Negro (*Giornale della R. Accademia di Medicinia dix Torino*, Luglio-Agosto, 1891, p. 484) made a communication before the Academy of Medicine of Turin on this subject.

Among the operative procedures employed in the treatment of certain forms of partial epilepsy, the excision of portions of the cerebral cortex corresponding to the motor centres has been of late successfully performed. This method of excision offers several inconveniences, the principal of which are the following:

1. Injury to the neighboring cortical or subcortical regions, thus producing paralysis or paresis of muscles not in the realm of the Jacksonian epilepsy.

2. These operations are very bloody, and sometimes give rise to serious hæmorrhages.

The author proposes to substitute for the knife, electricity, to accomplish the excision of the brain cortex. He places a large electrode, the positive pole upon the sternum of the patient, whilst with the negative pole tipped with platinum, which he inserts at various depths into the diseased brain substance, he effects electrolysis.

A current of two to three milliamperes will in a very short time destroy the diseased brain tissue.

An operation performed by Dr. Carle with this method in a case of meningo-encephalitic gummosa succeeded in suppressing the epileptic phenomena.

SODIUM HYPOSULPHITE AS AN INTESTINAL DISINFECTANT.

Goll says, in the *Pharmaceutische Centralhalle*, that the abandonment of sodium hyposulphite in treating diseases of the stomach and intestines for other and newer remedies is a great mistake. In solutions, according to Goll, it is far more efficacious than resorcin, Carlsbad water, and salts, and, in fact, any and all the newer remedies used in the treatment of gastric and intestinal catarrh. Especially does it seem indicated in chronic catarrh, and its effects in this direction are very marked.

PROPHYLACTIC TREATMENT OF POST-GONORRHŒIC AZOÖSPERMIA.

Dr. L. Seligmann (*La Sémaine Medicale*, No. 51, 1891) advises to treat every case of epididymitis as soon as the acute stage is passed by methodical massage, inunctions with ichthyol salve and permanent compression. At each séance one should massage besides the testicle, the epididymis, and finally the spermatic cord as high up as possible; then rub the parts with a salve of ichthyol (5 to 100 per cent.) and place them in a rubber suspensory. By these means the nutrition of the testicle is improved as well as that of the epididymis and spermatic cord; the bands and deposits of hyperplastic connective tissue, which cause contraction and obliteration of the tubes and seminiferous vessels and subsequent azoöspermia, are caused to soften and disappear.

HYOSCIN IN THE INSANE.

Dr. Willerup (*Hospitals-Tidende*, R. 3, Bd. 9, p. 389) has used hyoscin in the Oringe Asylum. The dose varied from one-half to three milligrammes ($\frac{1}{120}$ to $\frac{1}{20}$ of a grain) two times a day, with a maximum dose of six milligrammes ($\frac{1}{10}$ of a grain), given by the mouth. He finds it of service in the chronic insane, where there is great restlessness. It is a violent poison, and its action unfortunately inconstant.

MEDICINE.

CEREBRAL ABSCESS.

Dr. Carl Lohmeyer reports (*Ber. klin. Wochenschr.*, No. 37, 1891) a case of abscess of the brain in which cure resulted from operative treatment. Patient was a man, aged 47. Up to the age of ten he had been healthy, but then had scarlet fever. No history of discharge from either ear at any time. Early in 1862 an abscess formed behind and above the right ear; it discharged itself spontaneously, and healed. Precisely the same thing occurred a year later, and when he came under treatment (April, 1889) a scar was found at the part which had been affected. In March, 1889, he suddenly felt a boring pain on the right side of the head. This persisted up to the date at which he came under observation, becoming worse in the evening. While at work he experienced giddiness to such an extent that he had to hold on to some fixed object. He could not stoop without becoming giddy, but was never so whilst sitting or lying down. No vomiting at any time of the disorder. Patient's state, when first examined, was as follows: He looked very pale and ill, his muscles were weak, and his body was wasted. He complained that his sleep was broken by pain and dreams, the former of a dull, boring, persistent nature, and localized, the exact seat being at the upper extremity of a line, 7 centimetres in length, drawn vertically upwards from Merkel's horizontal skull line (this passes between the outer angle of the eye and the upper edge of the tragus), 1½ centimetre in front of the maxillary joint and the vertical line of Merkel running upwards from that point. Pain was also experienced ovér the right side of the head generally, and radiated towards the right eye and shoulder. The eye felt as if it were being forced out of its socket. At the seat of greatest pain there was much sensitiveness to pressure and percussion. Neither the skin nor the bone at this part presented any abnormality. There was bony depression about the size of a finger-tip upon the right parietal bone 3 centimetres behind and 4 centimetres above the posterior and upper margin of the pinna, and 9 centimetres from the point of greatest pain. The external table and the diploë were absent at this spot. The skin over it was covered with hair and adherent to the bone beneath. The ears were examined with negative result; power of hearing was normal on each side. Pupils, optic discs, and movements of eye muscles normal. Tongue covered with thick fur; its movements normal. Mastication occasioned pain in the head, especially at the tender spot before mentioned. Swallowing was attended by difficulty as regards solids. Speech slow but clear. Heart and lungs not apparently affected. Temperature, morning, 37.4° C.; evening, 38.4° C. Abdominal viscera normal. During the day the patient remained sitting in an apathetic state, but was still able to undress himself and get into bed unassisted.

His condition remained unaltered for about one month, at the end of which time œdematous swelling and paralysis of left leg were observed. During the next month (May) the feeling of giddiness left him, but the pains in the head increased, and the loss of sleep became more marked. He passed less urine. Pulse, 54 per minute, temperature as before. On May 10th he complained of creeping sensations and numbness of the left hand, also of a feeling of heaviness in the left arm. On the 16th paralysis of the left arm was complete; both left limbs were now paralyzed. Pulse, 40, temperature, unaltered. Three days later ptosis was observed (side not stated). Power of vision in right eye now became impaired, and an early stage of optic neuritis was discovered there; left eye normal; pupils dilated; they reacted slowly, especially the right pupil. Patient grew more apathetic, and somnolence took the place of sleeplessness. Speech became indistinct, and answers inappropriate. He could now only swallow water. Early in June he became quite comatose. Pulse 38, temperature 34.4°.

The author assigns as a cause of the abscess in the brain, which was diagnosed from the above symptoms, the caries which had affected the upper and back part of the right parietal bone in 1862, and had produced the scar and bony depression before mentioned. The abscess must have originated in the upper part of the lobus paracentralis, or anterior and posterior central gyri, and have spread downwards and forwards, having its middle point about the middle of the anterior central gyrus. On June 12th an incision 6 centimetres long was made across the point of greatest pain, in a direction from above downwards and backwards. A portion of bone rather larger than a shilling was removed, whereupon the dura mater (which was normal and did not pulsate) protruded. This was incised, and the brain substance, with convolutions flattened and œdematous, brought into view and incised in its turn, whereupon greenish-yellow pus squirted out. Between 60 and 70 g. of this was obtained. The abscess cavity was then washed out with tepid boracic solution under gentle pressure,

and subsequently filled loosely with iodoform gauze. A bandage impregnated with sublimate was next applied. On the outer wall of the abscess was found a warty growth the size of a pea, composed of detritus. This, the author says, is to be regarded as the efficient cause of the abscess. After the operation the patient slept. There was a slight increase in pulse rate, but no rise in temperature during the next two days. On the third day he began to regain consciousness. On the fifth and sixth days the optic disc became normal, and the paralysis disappeared in the reverse order to that in which it set in. The patient was discharged about five weeks after the date of admission, there being still a slight flow of pus from the wound. In six weeks and a half the abscess was completely healed and the external wound closed. The man resumed work. At the date of report —five months after operation—he was well, and had in the meantime worked daily.—*Brit. Méd. Jour.*

SURGERY.

FRACTURES OF THE LARYNX.

In an article based upon experimental researches upon fractures of the cricoid cartilage (*Gaz. Hebd.* No. 42, 1891), Dr. Zilgien, of Nancy, states that an ossified cartilage requires considerably less force to sustain a fracture, than does a cartilage which has simply undergone amiantaceous degeneration. This degeneration of hyaline cartilage precedes ossification and calcification, and is characterized by fine striations in the hyaline substance. If the hyaline structure is intact the cartilage cannot be broken. It bends, owing to its irresistible elasticity due to its perfect homogeneity and the thick layer of perichondrium in which it is enveloped.

Experiments were made upon the exposed larynges of cadavers, by exercising slow pressure with the aid of a dynamometer and gradually increasing the pressure until fracture was produced. The pressure requisite to fracture the thyroid cartilage averaged 22 to 28 kilogrammes. The line of fracture was most frequently median when the cartilages were ossified or calcified, and extra-median when they had merely undergone amiantaceous degeneration. This degeneration was less marked in the median line than laterally. The pressure necessary to break the cricoid cartilage averaged from 16 to 18 kilogrammes. It usually broke on the anterior arch in three places—one median and two lateral; sometimes there was a median fracture only, sometimes a median and one lateral; rarely two lateral without median fracture.

The cricoid broke, therefore, at the same points as in the living subject. Pressure with the thumb at the median portion of the cartilage produced first depression of the anterior arch; then, if the cartilage was affected with amiantaceous degeneration, a fracture was produced at the point of junction of the anterior arch with the chaton of the cartilage about 6 millimetres in front of the crico-thyroid articulation. It is, therefore, an indirect fracture, occurring near the extremities of the great transverse diameter formed by the flattening of the cartilage, and it takes place at its most feeble point. At the same moment that the lateral fracture takes place, the anterior arch breaks at the median line. If, however, the cartilage is ossified or calcified, the median fracture is produced first. If the pressure is made along the axis which unites the two crico-thyroid articulations, the fractures occur at the same points, but the median fracture always occurs first. The fractures follow the direction of the amiantaceous striations parallel to the line of fracture. Hence they sometimes resemble the surfaces of diarthrodial articulations, excavated on one fragment with corresponding prominence on the other.

The mucous membrane is rarely torn, usually only contused. The inter-crico-thyroid membrane usually remains intact. The perichondrium is almost always torn in front, while the posterior perichondrium remains intact in the vicinity of the lateral fractures.

TREPANATION FOR EPILEPSY

Professor Studsgaard, of Copenhagen, reports three cases of this operation. In the first case, that of a man aged 27, who, when 12 years old, had been exposed to an injury of the head, nothing abnormal was found in the dura mater or the brain; the operation only improved the condition of the patient slightly. In the other case, that of a young girl aged 17, whose injury was contracted seven years previously, a detached, flat, small bone was found lying between the dura and the bone, and was removed; recovery followed. The third case is still under treatment; the patient, a man aged 24, exhibited a depressed cicatrix over the left eye. The epileptic fits were evidently caused by bony productions (osteophytes) of a depressed part of the frontal bone.—*Nordiski medicinsk Arkiv*, vol. i, No. 18, 1891.

OBSTETRICS.

ACUTE OOPHORITIS COMPLICATING PREGNANCY.

Dr. Coe, writing on this subject in the *American Gynecological Journal*, December, 1891, gives several instances of such complication and says: "In a patient with a history of previous acute oöphoritis and peri-oöphoritis, whether occurring before or during pregnancy, apprehension must be felt as to the possibility of a recurrence of the former inflammation after delivery. Labor should be conducted with rigid aseptic precautions, and with the least possible amount of interference; rough manipulation of the fundus uteri, as in expression of the placenta, is especially to be avoided."

CRANIECTOMY.

Prengrueber (*Sém. Méd.*, January 27, 1892) has successfully operated on a boy, aged 9, suffering from simple idiocy with microcephalus. The opening, eleven cm. in length by two cm. wide, was made on the left side of the skull in the neighborhood of the left fronto-parietal suture. At this situation there was an exostosis both on the inner and external wall, the portion on the inside compressing the brain. Improvement was noted immediately after recovery from the anæsthetic, as his general appearance was better, his judgment increased, and his speech was more intelligible. On the next day the dribbling of saliva had ceased, and he was able to learn to blow his nose, a feat he had not accomplished before. He was soon able to play with his toys, and was much cleaner in his personal habits. Prengrueber considers that months and years must elapse before definite statements can be made in regard to permanent improvement.—*British Medical Journal*, February 6, 1892.

THE PUERPERIUM AS A FACTOR IN THE ÆTIOLOGY OF MULTIPLE NEURITIS AND DEGENERATION OF NERVE TISSUE.

H. Handford, M.D., in the *British Medical Journal*, Nov. 28, 1891, reports two cases of multiple nephritis, both probably of alcoholic origin, where the onset seemed most clearly to be determined by childbirth. He mentions a third case where the exact diagnosis was more difficult, but its onset immediately following confinement was equally clear. After quoting Grower's suggestion in this connection that "the tissue health is lowered, and hence slighter causes excite neuritis," he says the rapid progress of tabes dorsalis, and the frequency of optic atrophy in it when sexual relations are actively maintained, has been frequently noted. The relation of child-bearing is probably of a similar nature, and it is not suggested as a sufficient cause *per se*. On the other hand, child-bearing is a common occurrence, and the neuritis may have been a coincidence, but multiple neuritis is a comparatively rare disease, and for three out of six or seven cases, in both sexes, to commence with childbirth seems an undue proportion. The author then asks, "Is the influence of childbirth one of the factors which render alcoholic neuritis so much more common in women than in men, while drinking habits are more prevalent in the latter?"

GYNÆCOLOGY.

SEPTICÆMIA FROM PUTRID FŒTUS.

Gendron (*Arch de Tocol, et de Gynec.*, October, 1891,) read a clinical report of two cases before the Obstetrical Society of Paris on October 8th. A woman had septicæmia close upon term, with high temperature and other grave symptoms. Labor set in, and when the os was half dilated, Gendron hastened the dilating process with his hand and extracted the decomposed fœtus. After rigorous antiseptic treatment during childbed, the patient recovered. A similar case was recently under his care. The cervix was rigid and dilatation imperfect. Gendron made an incision into the os posteriorly; then dilatation was easily completed and the putrid fœtus extracted. Recovery was perfect after the same treatment as in the first case.—*Brit. Med. Jour.*

HYDATIDIFORM MOLE.

Lwow (*Centralb f. Gynak.*, No. 1, 1892) has had four molar pregnancies under his own observation. The most evident of the predisposing causes is hyperplastic endometritis. There was no evidence of syphilis in any of the cases. All four had previously been under his care for uterine disease—endometritis, retroflexion, dysmenorrhœa, etc. Three were multiparæ, the fourth was a primipara. In all, diagnosis of molar pregnancy was easy on account of the unusually rapid increase of size of the uterus, and the free flooding; whilst in two cases the grape-like masses could be felt through the os uteri. In two cases the mole was spontaneously discharged. In the others it had to be removed with the hand, as repeated hæm-

orrhages threatened the patient's life. The entire mass could not be removed in one instance, as Lwow feared the uterine walls, as thin as paper, might be perforated by the finger. After the greater part of the mole had been taken away, the uterus contracted freely, and the remainder came away during convalescence. Lwow holds that warm irrigations, thrown up into the uterus, are perfectly justifiable after molar delivery. In all four cases they were employed, and proved satisfactory.—*Brit. Med. Jour.*

PELVIC INFLAMMATION IN WOMEN.

Dr. Potter, in a pathological study of this most interesting condition, which furnishes such a large proportion of the gynecologist's work, regards McMurtry's grouping of varieties of inflammation as about the best. It is as follows: 1. Inflammation of serous and cellular intra-pelvic tissues cannot be separated clinically nor histologically, hence they cannot be properly distinguished by the terms perimetritis and parametritis. 2. The pelvic cellulitis of Emmet, which corresponds to the peri-uterine phlegmon of Nonat, is as rare as inflammation of the cellular tissue elsewhere. 3. Pelvic inflammation is, generally speaking, peritonitis resulting from disease of the ovaries or Fallopian tubes, or both. 4. Pelvic peritonitis presents every grade of activity, and is always symptomatic, never idiopathic. These inflammations are either of puerperal, gonorrhœal, or miscellaneous origin, and under the latter are included all infections of the endometrium by instruments, medicinal agents, and traumatism.—*American Gynecological Journal*.

PÆDIATRICS.

ZONA IN CHILDREN.

According to Comby (*La Bulletin Médical*, November, 22, 1891), the pathogenesis of herpes remains to be explained. He has studied the disease during eight years, and has collected thirty-three cases, twelve of which occurred in boys, and twenty-one in girls. The predominance of the disorder in the female sex has been observed by other authors. According to the statistics of Comby, the disease occurred in four children of less than two years of age; the ages of the rest of the cases varied from two to ten years. One of the patients was only eight months old. The affection did not present a contagious character, and it was more common in the spring and summer than in the months of autumn and winter. The symptoms are purely of an objective character, and it is worthy of note that, in children, herpes assumes a benign type. There is generally a slight febrile reaction, accompanied with gastric disturbance. The neuralgic pains are much less troublesome than in the case of adults. The author believes the disease to be essentially an infectious neuritis, and that the characteristic eruption is nothing more than a trophic manifestation which is proportionate to the extent of the lesion. Herpes may have also, however, a traumatic or other origin. The treatment is quite simple, and consists in the local application over the diseased parts of antiseptic remedies.

TREATMENT OF DIPHTHERIA BY IRRIGATIONS OF SALICYLIC ACID.

Parisot highly recommends (*Bull. Gén. de Thérap.*) the employment of irrigations of salicylic acid (1 to 1000) in diphtheria, as proposed originally by Weise and others, and affirm that whereas, before resorting to this method of treatment, the mortality from that disease as occurring in his practice was large—ten cases out of every fourteen— in a recent epidemic in which he had relied on the irrigations, there were only five fatal cases out of every twenty-four. He makes the solution by dissolving 1 part of the acid in twenty parts of alcohol, and then adding 980 parts of water. The apparatus which he used for the irrigation was simply a fountain syringe, the rubber tubing ending in a small glass tube. The head of the child is held by an assistant, the tongue depressed, and the jet directed into the mouth and posterior pharynx with sufficient force to detach and remove the false membranes if they happen to be loose. The quantity used must depend on the judgment of the operator. The irrigation does not wholly replace other remedial measures, such as the frequent administration of stimulants. Parisot makes some remarks as to the action of salicylic on false membranes which, if true, are of great practical importance. He believes he ascertained by experiment that this acid is destructive to diphtheric formations; in distilled water the false membrane was simply disaggregated, and this disaggregation took place slowly, while in solutions of different strengths of salicylic acid the exudate disappeared rapidly; at the end of a few minutes nothing was found but the meshes of the network serving for support to the cells of the exudation. The stronger the solution of salicylic acid the more prompt and complete was the disappearance of the exudate.

HYGIENE.

"L'ESTOMAC ET LE CORSET."

Dr. Chapotot, in a recent essay published by Baillière under this title, gives a fair summary of the opinions of experts such as Bouvier, Dickinson, Sibson, and others, on the real and imaginary evils attributed to constriction of the waist. The question of the true position of the stomach is very important in respect to the correct interpretation of abnormal relations of the abdominal viscera detected after death. The most original portion of Dr. Chapatot's essay refers to a matter of some interest to ladies. Young women have often reason to complain of disagreeable noises caused by air moving about in the epigastric region. Our author attributes these noises, which differ from borborygmi, to a vertical bilobulation of the stomach caused by the pressure of stays. During expiration the upper lobe is relieved of pressure by the ascent of the diaphragm. The lower lobe is, on the other hand, subjected to great pressure from the abdominal muscles. Hence air and liquids are forced upwards into the upper lobe through the narrow isthmus produced by the pressure of the stays; as they pass through the isthmus and issue out of it the characteristic gurgling sound is produced. If the stays be taken off, the sounds are no longer heard, but they may be reproduced by applying any other form of constriction to the abdomen at the same level.—*Brit. Med. Jour.*

A CHEAP DISINFECTANT.

At this season of the year and during the summer and autumnal months, disinfectants should be kept ready at hand for needed use, but not in any measure to take the place of cleanliness.

The nitrate of lead is the cheapest disinfectant known that fulfils its intent. It does not, however, prevent putrefaction. The chloride of lead is much more effective in all directions. It is made by dissolving a small teaspoonful of nitrate of lead in a pint of boiling water; then dissolve two full teaspoonfuls of common salt in eight quarts of water. When both are thoroughly dissolved, pour the two mixtures together, and when the sediment has settled you have two gallons of clear fluid, which is the saturated solution of the chloride of lead. A pound of nitrate will make several barrels of the liquid. The nitrate of lead costs from eighteen to twenty-five cents a pound at retail.—*Monthly Bulletin.*

THE INFLUENCE OF INFUENZA ON THE DEATH-RATE OF 1890.

The report of the Registrar-General for 1890, relating to the births, marriages, and deaths in England and Wales during that year has just been issued. The birth-rate, which had shown a continuous decline since 1876, was the lowest recorded during any of the last fifty years, although the marriage-rate during the last four years has shown a steady increase, and was higher in 1890 than in any year since 1883. With regard to the deaths during the period under review, it appears that the standard of public health reached in recent years was not maintained during 1890. The death-rate, which had been 19.1, 18.1, and 18.2 per 1,000 persons living in the preceding three years, 1887-88-89, rose to 19.5 in 1890. It should be noted, nowever, that compared with other earlier years, this death-rate was still a low one; for in none of the ten years 1881-90 was the death-rate as high as 20.0 per 1,000, whereas in no year previous to 1881 was it ever so low. The increase in the rate of mortality in 1890 is attributable to the epidemic of influenza, to which, although it was generally prevalent only during the first quarter of the year, 4,523 deaths were directly ascribed. This, however, probably represents but a small proportion of the mortality really attributable to influenza; for the immense increase in the mortality from respiratory diseases at the same time, notwithstanding the mildness of the season, was clearly due to the same cause. The Registrar-General estimates that the number of deaths directly or indirectly due to epidemic influenza in 1890 at upwards of 27,000, which is equivalent to a death-rate of almost one per 1,000. It is satisfactory to note that the mortality from nearly all other zymotic diseases was below the average. Only 16 deaths resulted from small-pox in 1890, the smallest number in any year on record. The deaths referred to constitutional diseases, especially to phthisis, showed a marked increase, probably attributable to the fact that a number of persons of phthisical constitution were prematurely carried off by the influenza epidemic during the earlier part of the year.—*Brit. Med. Jour.*

ANOTHER REMEDY FOR WHOOPING-COUGH.

M. Chibert declares that he has rapidly stopped the kinks of whooping-cough by sprinkling finely powdered iodoform on the child's pillow—*Répertoire de Pharmacie*, No. 8, 1891.

MEDICAL CHEMISTRY.

TESTING LARD.

The following method for detecting vegetable oils in lard is suggested by Welmans: Dissolve the lard in chloroform, and shake the liquid with a solution of sodium molybdate in nitric acid. If the lard is pure the color of the mixture remains unchanged, while if vegetable oils are present the molybdic solution is reduced and assumes a green color, which is the more intense the more of the vegetable oil is present. On supersaturating this liquid with ammonia the green color changes to blue, while with pure lard no color is developed even after the addition of the ammonia.—*Zeitsch. f. angew. Chem.*

METHYL BLUE AND METHYLENE BLUE.

Both of the substances mentioned in the title have been introduced into medicine, but we have reason to know that the two substances are not only constantly confounded by wholesale druggists and dispensing pharmacists, but that the impure commercial articles are still kept in stock by many and dispensed.

Methyl blue has been recommended as an antiseptic in diphtheria and certain other diseases. Dr. H. Beyer (of Staten Island), in a paper published in the *Medical News*, first proposed it for this purpose, and recommended to apply it in form of a mixture containing 2 parts of methyl blue and 98 parts of powdered sugar.

Methylene blue is given internally, in doses of about 3 grains (in pills or capsules), in rheumatism of the joints and muscles; also as an analgesic, administered hypodermically in doses of $\frac{1}{16}$ to 3 grains; and has recently been lauded as an antimalarial remedy in doses of $1\frac{1}{2}$ grains, the daily doses not to exceed 10 grains.

Methyl Blue is the sodium salt of triphenyl-pararosaniline-sulphonic acid. It is also known as Methylblue M B I for cotton, and in German also under other names. It constitutes a dark-blue powder, soluble in water, forming a blue solution. The latter is not altered by hydrochloric acid. Addition of soda renders the solution reddish brown. In concentrated sulphuric acid, methyl blue dissolves with a reddish-blue color, which turns to blue upon dilution with water.

Methylene Blue, also known as Æthylenblau, Methyleneblue D B B, is a salt of tetramethylthionine (a body classed with the so-called thiazines, which are sulphurated derivatives of the aniline series). The most usual salt met with in the market is the double chloride of *zinc* and tetramethylthionine. According to Schultz (*Chemie des Steinkohlentheers*, 2, 762), the simple hydrochlorate of the base is also put on the market under the name of methylene blue. But we have so far not encountred the latter, all the samples which have come under our notice being the *zinc* salt, leaving on combustion about 10 per cent. of zinc oxide. It is possible that a zinc-free methylene blue is now on the market, but, as we have not had any occasion to examine the article for some months, we cannot be sure. At all events, it behooves those who use methylene blue internally to know whether they are given a zinc salt or not. Hence every fresh supply should be examined for zinc, which is easily accomplished by igniting a small weighed quantity in a crucible (which may be much facilitated by moistening the carbonized and cooled mass occasionally with a little water and nitric acid) until all signs of carbon have been burned off. The residue, if consisting of zinc oxide, will give the appropriate reactions.

We are unable to say whether the first proposers of methylene blue as an internal remedy were aware of the presence of zinc in the commercial article, or whether they used a zinc-free product. At all events, we are aware that many physicians who used the substance in this country employed the commercial article containing zinc. The question is now, What difference in therapeutic action is there between the two products, that comtaining zinc and that free from it? And did the beneficial effects of the remedy depend on the organic coloring matter exclusively, or on its combination with zinc? This is a problem to be solved by the pharmacologists.

HŒMOL AND HŒMOGALLOL.

There are two preparations containing iron, which for assimilation far surpass any compound previously used; they were first prepared by Prof. Kobert by the action of reducing agents upon the blood-coloring matter; in the first-mentioned zinc-dust is used (a minute quantity of zinc is allowed to remain in this preparation as it exerts a favorable action in certain defects of the stomach and intestines): in the last mentioned, pyrogallol is used; the former is a dark brown, the latter a red brown powder. The dose ranges from 0.1–0.5 gm. three times a day; an agreeable form of administration was found in chocolate tablets, each representing

a little over one milligram metallic iron, one to be taken fifteen minutes before meals. Patents have been applied for, for these two preparations.—*Oesterr. Ztschr. f. Pharm.*, 1891, 724.

NEWS AND MISCELLANY.

DANGEROUS PETS.

A French scientist declares that the domestic pets of the world carry at least thirty per cent. of the common contagious diseases from house to house.

One of our trained nurses sent to a physician of her acquaintance, of this city, under whom she had served, for a motto for her calendar. He sent her the following improvisation:

Instil hope,
Don't mope,
Use Soap,
So you'll best with evil cope.

THE DEBATE ON PHAGOCYTOSIS.

The debate at the Pathological Society came to a close on Tuesday last, having thus occupied three evenings. It has certainly been of great value, and it says much for the skill with which it has been conducted that the interest in a subject which involves of necessity points and arguments that are not easily grasped by the uninitiated should have been so well maintained. The paper with which Dr. Ruffer opened lasts Tuesday's proceedings was well worthy of the subject, and of one who is entitled to be regarded as perhaps the foremost student of phagocytosis in this country. It seemed to leave no debatable point untouched, and dealt in masterly fashion with the criticisms of Dr. Klein and the alternative hypothesis of Buchner and his followers. One great merit of this paper was the care with which its author avoided using the somewhat equivocal phraseology that has sprung up around this subject. There is no such thing, he boldly said, as "immunity" in the absolute sense, and throughout he preferred to speak of animals as being "naturally resistant" or "artifically protected," instead of using the hyperbolic terms that have become current. Phagocytosis, he maintained, is the only process which has been proved to be essential in the production of this resistance or protection, although "neither Metschnikoff nor any of his pupils has ever said that this was the *only* cause of natural or artificial resistance." Mr. Ballance's lucid exposition of the active part played by the fixed tissue cells in inflammation and repair and the subordinate *rôle* of leucocytes led up to the declaration that the former are probably the chief agents in phagocytosis; although the essentially chemical nature of the processes whereby immunity is secured was also pointed out. Mr. Hankin's interesting discovery of a proteid in the spleen of the white rat having bactericidal properties—although discounted by the supporters of the exclusive phagocytosis doctrine—undoubtedly gives strong support to the chemical theory of protection, and his speech was interesting as affording a reply to some of the arguments of Dr. Ruffer. In replying on the whole case, Dr. Sims Woodhead claimed that all the evidence hitherto adduced spoke in favor of phagocytosis being the agency at work in the living body, since the evidence on behalf of the humoralistic doctrine was derived from blood-serum or its products, which probably do not exist as such in the living animal. The summoning up of Sir George Humphry was weighty and judicial, and his tribute to English bacteriologists was felt to be well deserved. The debate will, we doubt not, go far towards opening up new paths of research in this extremely interesting question.—*Lancet.*

MEDICAL PROVERBS.

(*German.*)

A physician is an angel when employed, but a devil when you must pay him.

Dear physic always does good.

A disobedient patient makes an unfeeling physician.

(*Spanish.*)

What cures Sancho makes Martha sick.

The earth hides as it takes
The physician's mistakes.

He that sits with his back to a draft, sits with his face to a coffin.

Of the malady a man fears, he dies.

He that would be healthy must wear his winter clothes in summer.

(*English.*)

Diseases are a tax upon our pleasures.

A good surgeon must have an eagle's eye, a lion's heart and a lady's hand.

Tender surgeons make foul wounds.

(*French.*)

A physician is a man who pours drugs, of which he knows little, into a body of which he knows less.